A DISSERTATION ON

A PROFILE OF EPILEPSY & EPILEPTIC SYNDROMES IN CHILDREN LESS THAN 12 YEARS

MD BRANCH (VII)

PEDIATRIC MEDICINE

THE TAMILNADU

DR.M.G.R. MEDICAL UNIVERSITY

CHENNAI, TAMILNADU

SEPTEMBER 2006

CERTIFICATE

This is to certify that this dissertation entitled "A PROFILE OF EPILEPSY & EPILEPTIC SYNDROMES IN CHILDREN LESS THAN 12 YEARS" submitted by DR. M. SARAVANAN to the faculty of Pediatrics, The Tamil Nadu Dr. M.G.R. Medical University, Chennai, in partial fulfillment of the requirement of the award of M.D. Degree Branch VII (Pediatric Medicine) is a bonafide research work carried out by him under our direct supervision and guidance.

DR. M.L. VASANTHAKUMARI. M.D. D.C.H.,
PROFESSOR & H.O.D.

INSTITUTE OF CHILD HEALTH & RESEARCH CENTRE,
GOVT. RAJAJI HOSPITAL &
MADURAI MEDICAL COLLEGE
MADURAI.

ACKNOWLEDGEMENT

I am greatly indebted to my teacher **Dr.D.Meikandan MD DCH DM (Neuro)** *Additional Professor of Paediatrics* for his supervision, guidance and encouragement While undertaking this study.

I am also extremely grateful to **Prof. Dr. M.L. Vasantha Kumari,** Professor and Head of the Department of Paediatrics, Government Rajaji Hospital and Maduri Medical College, Madurai For his guidance, which has helped me in completing this work successfully.

I would like to thank **Prof. Dr.P. Amutharajeswari**, *our unit Chief, Additional Professor of Paediatrics* who encouraged me to undertake this study and guided me to a great extent.

I also thank all the members of the Ethics Committee for their valuable suggestions. I thank profusely our *Assistant Professors* **Dr. K.Mathiarasan** and **Dr. M.Balasubramanian** for their critical review and analysis at every stage of this study.

I thank the **Dean**, Government Rajaji Hospital and Madurai Medical College, Madurai for permitting me to use the clinical materials and for taking free CT for some very poor children.

CONTENTS

INTRODUCTION

DEFINITIONS

Seizure (convulsion) is defined as a paroxysmal involuntary disturbance of brain function that may manifest as an impairment or loss of consciousness, abnormal motor activity, behavioural abnormalities, sensory disturbances or autonomic dysfunction. Some seizures are characterised by abnormal movements without loss or impairment of consciousness.

Epilepsy is defined as recurrent seizures unrelated to fever or due to an acute cerebral insult. Seizures may be either generalized or focal. In generalized seizures the electrical discharge originates in midline diencephalic areas.

In focal seizures the discharge arises in a localized area of the cortex ; it may remain circumscribed or spread to adjacent cortical regions or to the opposite hemisphere via the corpus collosum and may become generalized.

Epilepsy is a common neurological disorder in childhood; according for about 70% of all neurological disorders, further more 70% of all epilepsies have their onset in childhood and adolescence. Because of the magnitude of the problem, the primary care physician should be

familiar with the spectrum of childhood seizures and epileptic syndromes.

CLASSIFICATION OF EPILEPTIC SEIZURES:

Classification of the epilepsies is important not only in clinical practice but also in epidemiology, because the etiology, treatment and prognosis remarkably with the type of epilepsy. Three decades ago epilepsies, based on clinical observations, were classified simply as grand mal, petit mal, and psychomotor. The first classification of seizures to be internationally accepted, the International Classification of Epileptic Seizures (ICES) was published in 1970.[1]

One of the most important contribution of this classification was subdivision of seizures into those with a focal onset or partial seizures and those in which no localization of onset was possible, i.e., generalized seizures. Interictal and ictal EEG, age, anatomical substrate and etiology were correlated with clinical events. Advent of long-term EEG and simultaneous video EEG monitoring has greatly increased the descriptive accuracy of seizure phenomena. The result was revision of the ICES in 1981 .[2] The revised classification was based on clinical semiology, interictal EEG findings, and ictal EEG patterns. Data concerning age, anatomical substrate and etiology were excluded. The revised ICES subdivides partial seizures into simple (without loss of

consciousness) and complex (with loss of consciousness) and secondarily generalized.

ILAE revised classification of epileptic seizures (1981)

Partial (focal, local) seizures

Simple partial seizures

1. with motor signs: focal, motor, jacksonian, versive, postual, phonatory
2. with autonomic symptoms and signs.
3. With somatosensory or special sensory symptoms (simple hallucinations, olfactory, gustatory. vertiginous.
4. With psychic symptoms (disturbances of higher cerebel functions): dysphasic, dysmnesic, cognitive, affective, illusions, strucetered hallucinations

Complex partial seizures (with impairment of consciousness)

1. Simple partial onset of followed by impairment of consciousness
2. With simple partial features (A1-A4) followed by impaired consciousness
3. With impairment of consciousness at onset with features (A1-A4) with automatisms.

Partial seizures evolving to secondarily generalized tonic-clonic seizures (GTC)

1. Simple partial seizures evolving to GTC
2. Complex partial seizures evolving to GTC
3. Simple partial seizures evolving to complex partial seizures, evolving to GTC

Generalized seizures

- Atonic seizures Absence seizures, with impairment of consciousness, with clonic, atonic, tonic or autonomic components, or with automatisms occurring alone or in combination.
- Atypical absences, more pronounced changes of tone than in absence seizures; onset and/or cessation not abrupt.

  Myoclonic seizures (single or multiple)

Clonic seizures

Tonic seizures

Tonic-clonic seizures

Unclassified

Neonatal seizures and others

In 1985 the International League Against Epilepsy (ILAE) proposed the International Classification of Epilepsies and Epileptic Syndromes (ICEES) and refined it in 1989 taking into consideration

various newly described epilepsies and epileptic syndromes . Epileptic syndrome is defined as “cluster of signs and symptoms customarily occurring together”. In addition to seizure type, epilepsy syndrome classification considers are of onset, intellectual development, neurological findings, anatomical substrate, and possible etiology. Many of these syndromes, however, may have different etiologies in different patients, and so, with few exceptions, epilepsies have not been defined as specific diseases. This classification recognizes the dichotomy between partial and generalized seizures. The term “localization related” is used instead of partial or focal to recognize that some types of seizures have a shifting focus. The syndromic classification also establishes a second dichotomy between age related idiopathic epilepsies on one hand and symptomatic and cryptogenic epilepsies on the other. Symptomatic indicates that the epilepsy has a known or suspected etiology. Cryptogenic differs from symptomatic only in that the cause is unclear. Idiopathic acquired a clear connotation of an age-related onset and a genetic etiology. In children with symptomatic generalized epileptic syndromes, seizures are either the initial, identifying symptom or the most important feature. These diseases include major cerebral malformations, storage disorders, and various inborn errors of metabolism.

ILAE 1989 Classification of epilepsies and epileptic syndromes.

1. **Localization-related (focal, local, partial) Epilepsies and Epileptic Syndromes**

1.1 Idiopathic (with age-related onset)

- Benign childhood epilepsy with centrotemporal spikes
- Childhood epilepsy with occipital paroxysms
- Primary reading epilepsy

1.2. Symptomatic

- Chronic progressive epilepsia partialis continua of childhood (Kojewnikow syndrome)
- Syndromes characterized by seizures with specific modes of precipitation
- Temporal lobe epilepsies
- Frontal lobe epilepsies
- Parietal lobe epilepsies
- Occipital lobe epilepsies

1.3 **Cryptogenic**

2. Generalized Epilepsies and Syndromes

2.1 Idiopathic (with age-related onset)

- Benign neonatal familial convulsions
- Benign neonatal convulsions

- Benign myoclonic epilepsy in infancy
- Childhood absence epilepsy (pyknolepsy)
- Juvenile absence epilepsy
- Epilepsy with grand mal seizures on awakening
- Other generalized epilepsies (not defined above)
- Epilepsies with seizures precipitated by specific modes of activation

2.2 Cryptogenic or Symptomatic

- West syndrome (infantile spasms, Blitz-Nick-Salaam Krampfe)
- Lennox –Gastaut syndrome
- Epilepsy with myoclonic-astatic seizures
- Epilepsy with myoclonoc absences

2.3 Symptomatic

2.3.1 Nonspecific etiology

- Early myoclonic encephalopathy
- Early infantile epileptic encephalopathy with suppression-brust EEG
- Other symptomatic generalized epilepsies not defined above.

2.3.2. Specific syndromes (diseases in which seizures are a presenting or predominant feature)

3. Epilepsies and Epileptic Syndromes Undetermined whether Focal or Generalised.

3.1 ***with both generalized and focal seizures***

- Neonatal seizures
- Severe myoclonic epilepsy in infancy
- Epilepsy with continuous spike –waves during slow-wave sleep
- Acquired epileptic aphasia (Landau-Kleffner syndrome)
- Other Undetermined epilepsies not defined above.

4.Special syndromes

4.1. Situation- related seizures

- Febrile convulsions
- Isolated seizures or isolated status epilepticus
- Seizures occurring only when there is an acute metabolic or toxic event.

CHILDHOOD EPILEPSIES AND EPILEPTIC SYNDROMES

Epileptic syndromes in childhood have different clinical expression and prognosis. The clinical picture ranges from quite benign to extremely severe epilepsies with associated handicaps, ranging from most benign with recovery to the most severe with developmental regression.[5,6] The ideal practical approach to childhood epileptic syndromes is to classify them according to age of onset as shown below.

Infancy

Febrile Seizures

Febrile seizures are the most frequent type of seizures in infants and preschool children and occur in 3-4% of children in this age group. Family history of febrile seizures is noted in 40% of children. Typical febrile seizures occur between 6 months to 6 years of age. Febrile seizures occurring before 6 months of age should always raise the suspicion cause. Seizures are described as

Classification of epilepsy syndromes according to usual age at onset.

Neonatal period

Benign neonatal convulsions

Benign neonatal familial convulsions

Miscellaneous neonatal seizures

Infancy

Febrile seizures

Early infantile epileptic encephalopathy

Early myoclonic encephalopathy

Infantile spasm (West Syndrome)

Severe myoclonic epilepsy of infancy

Benign myoclonic epilepsy of infancy

Benign partial epilepsy of infancy

Benign infantile familial convulsions

Symptomatic/cryptogenic partial epilepsies

Early childhood (toddler and preschool age)

Epilepsy with myoclonic absences

Lennox-Gastaut syndrome

Epilepsy with myoclonic-astatic seizures (Doose syndrome)

Acquired epileptic aphasia (Landau-Kleffner syndrome)

Epilepsy with continuous spike-waves during slow-wave sleep

Epilepsy with gelastic seizures and hypothalamic hamartoma

Symptomatic/cryptogenic partial epilepsies

Childhood (school age), adolescence, and young adulthood

Childhood absence epilepsy

Benign partial epilepsy with centrotemporal spikes

Benign occipital epilepsy

Childhood Grand mal syndrome

Reflex epilepsies (e.g. photosensitive epilepsy, reading epilepsy)

Juvenile absence epilepsy

Epilepsy with tonic-clonic seizures on awakening

Juvenile myoclonic epilepsy

Autosomal dominant nocturnal frontal lobe epilepsy

Symptomatic/cryptogenic partial epilepsy

tonic, clonic, or tonic-clonic events lasting less than 15 minutes and often occur within 24-38 hours of fever. Rarely, status can be the feature. Seizures recur in one-third of cases. Children with typical febrile seizures have good outcome and the risk of epilepsy is 1% Atypical febrile seizures may be partial, lasting for more than 15 minutes, and can be frequent. Children with atypical febrile seizures may have developmental delay and family history of epilepsy. The risk of epilepsy is 10%. EEG and neuroimaging may be abnormal.[7]

Management includes control of temperature with antipyretics and treatment of the cause of fever. Rectal diazepam or lorazepam can be used to abort the seizure. Long-term AED therapy is not indicated and Phenobarbital theraby is associated with significant risk of neurobehavioral side effects.

Early Infantile Epileptic Encephalopathy

Early infantile encephalopathy (EIEE), also known as Ohtahara syndrome, [8] is charactereized by onset in early infancy with very frequent generalized tonic and partial seizures. EEG shows burst suppression pattern. Severe structural abnormalities like dentate-olivary dysplasia[9] are common. Although EEG abnormalities disappear within one year, development remains very poor and seizures remain intractable. AEDs are not effective. Surgery may be helpful in patients with focal cortical dysplasia. [10] Some Children may evolve into West syndrome or LGS with neuronal maturation.

Infantile Spasms- West Syndrome

The onset of infantile spasms or West syndrome (WS) commonly occurs between 3 and 12 months of age in most patients. [11] Late onset WS is increasingly recognized. [12] It consists of a triad of infantile spasms which are commonly flexor, occurring in clusters, increasing upon waking, sometimes with focal features, mental retardation and hypsarryhthmia (typical or modified) seen on EEG. In symptomatic WS,

development is delayed from birth while in cryptogenic, psychomotor retardation occurs after the onset of seizures.

Perinatal insult, tuberous sclerosis and cerebral malformations are the most common causes. Normal neurological outcome is reported only in 10%-15% of cases. Prognosis is poor in symptomatic WS, when onset is early within 3 months of life and when other seizure types are associated. Some of the cases of WS evolve into LGS later. Response to treatment is better when treatment is started early with either ACTH or oral steroids. Vigabatrin has been found to be every useful symptomatic WS, secondary to tuberous sclerosis. [13] However, usual field defects reported with vigabatrin cannot be tested in this age group. Surgery can be considered in cases where WS is secondary to focal cortical plasia. [14] For a single large tuber can be removed in tuberous sclerosis.

Severe Myoclonic Epilepsy of Infancy

Severe myoconic epilepsy can occur in previously healthy infants, more often boys than girl. A family history of febrile seizures or epilepsy is common (55%-60%). During the first year of life, unilateral or bilateral clonic or tonic –clonic seizures occur mostly in febrile context, later nonfebrile seizures rapidly develop. Generalized or fragmented erratic myoclonias are found between the ages of 1 and 4 years. Other seizure types include atypical absences and partial seizures. Psychomotor development is retarded and patients develop ataxia and

pyramical signs. EEG shows bursts of genearalized spike and wave charges. Therapy is difficult and prognosis is unfavorable. [15]

Benign Myoclonic Epilepsy of Infancy

Benign myoclonic epilepsy of infancy (BMEI) is characterized by its onset between 6 months to 3 years. It is more common in males than females. family history of epilepsy is reported in 30%. Frequent massive myoclonus is the most common seizure type. Development remains normal. EEG shows generalized spike and wave complexes and clinical expression with normal background. Response to valporate is excellent if started early. GTCS may occur later in adolescence. [16]

Benign partial Epilepsy of Infancy

Benign partial epilepsy of infancy (BPEI) occurs in infants from 3 months to 6 months. There is family history of aferbrile seizures in infancy in 50% of seizures. Seizures are focal and may generalize and last for few days, involving the side or the other. Ictal EEG shows rhythmic activity over parietal, temporal occipital regions. Interictal EEG remains normal. Outcome is favorable.

Early Childhood

Epilepsy absence described by Tassinari [17] has its onset between 1-12yr. of with a male preponderance and family history of epilepsy in 19% of the cases. Seizure semiology is characterized by absence with very frequent severe bilateral rhythmic myoclonus often with tonic

contraction. There is developmental delay in 44% cases. The condition is resistant to drugs with unfavorable outcome. Ictal EEG is always associated with generalized bisynchronous, 3 Hz spike and wave complexes.

Lennox- Gastaut Syndrome

Lennox-Gastaut syndrome (LGS) [18] is characterized by multiple seizure types. Axial tonic seizures are the hallmark of LGS. Later on atypical absence, atonic and myoclonic falls and recurrent status may be observed. Cognitive and behavioral abnormalities are common., Age of onset is 1-8 yrs. LGS may be idiopathic or symptomatic. History of WS can be present in 25% of the cases. The awake EEG record shows 2-to-2.5-Hz spike-and-wave and polyspike-and-wave discharges, which are usually diffuse and maximal bifrontally. Prognosis is unfavorable and only a minority of patients achieves seizure control. Newer antiepileptic drugs (AEDs) like felbamate; lamotrigene and topiramate have been found to be useful. [19] Anterior corpus callosotomy may reduce seizure frequency in some patients. Astatic seizures preceded by a prominent tonic component are most improved by these procedures in some cases. [20]

Myoclonic Astalic Seizures

Myoclonic astatic seizures (MAS) described by Doose14 has its age of onset 1-4 yr., occurring more frequently in male. A strong genetic

component is suggested by history of epilepsy or EEG abnormality in 32% of first-and second- degree relatives. Development is normal in majority of the cases before onset of seizures. Myoclonic, astatic or myoclonic astatic seizures characterize the syndrome. Short absences or generalized tonic clonic convulsions may be associated but there is no tonic component as in LGS. EEG shows generalized spike-and-wave discharges, photosensitivity and 4-7 Hz rhythmic. Outcome is variable with either spontaneous cure or malignant course. Valproate, ACTH or oral steroids, ethosuximide, and acetazolamide have been found to be useful. Sometimes it is difficult to differentiate clinically MAS from cryptogenic WS, BMEI or idiopathic LGS. Prolonged follow up with serial EEGS and response to treatment may help in the diagnosis.

Acquired Epileptic Aphasia – Landau Kleffner Syndrome

Landau-Kleffner syndrome (LKS) is characterized by onset during early childhood with acquired aphasia starting with auditory verbal agnosia, hyperkinesias, personality disturbances, severe autistic-type behavior etc. There may not be any seizures or they may be heterogeneous –GTCS, complex partial or atypical absence. EEG is nonspecific or may show multifocal spike-wave discharges or electrical status during sleep. Development is normal. Prognosis is variable, 40-50% of patients may be able to lead normal socioprofessional life.

Effect of AEDs on aphasia is doubtful. High-dose steroids may be helpful. Multiple supial transaction of perisylvian cortex may be useful in selected cases. [21]

Epilepsy with Continuous Spikes and Waves during Slow Sleep

Epilepsy with continuous spikes-and-waves during slow sleep (CSWS) [22] has its onset between 8-12 years. It may be either symptomatic or idiopathic. Seizures can be of any type: GTCS, orofacial, generalized clonic, typical or atypical absence, tonic, atonic or rarely myoclonic. Awake EEG shows diffuse 2-3 Hz spike-and-wave discharges with focal features predominantly over frontal and centrotemporal regions. During sleep, there is continuous, bilateral, diffuse slow spike-and-wave discharges occupying 85-100% of the recording and the discharges disappear during REM sleep. Neuropsychological sequelae are common. Prognosis is not favorable and the behavioral disorders associated with CSWS are often difficult to treat, even though seizures disappear and EEG becomes normal.

Epilepsy with Gelastic Seizures and Hypothalamic Hamartoma

Hypothalamic hamartomas, although not arising from cerebral hemispheres are responsible for well defined syndrome with age of onset around 2 years, characterized by frequent giggling seizures. [23] psychomotor retardation occurs later, often associated with other types

of seizures- GTCS, complex partial. Precocious puberty is common. Diagnosis is confirmed on neuroimaging. Seizures remain intractable. Surgery is difficult but can improve the seizures. [24].

Childhood

Childhood Absence Epilepsy

Childhood absence epilepsy(CAE) [25] occurs between 3-12 years. Family history of absence and GTCS may be seen in 15-40% of cases. Seizures are characterized by typical absences. which may be simple in 10% or complex, associated with mild, clonictonic, atonic, myoclonic or automatisms in 90% of cases. [26] Seizures are precipitated by hyperventilation in almost all cases. EEG typically shows generalized, bisynchronous, symmetrical 3 Hz spike-and-wave discharges with abrupt onset and termination and background activity is normal. Prognosis is favorable. Both ethosuximide and valproate suppress absence seizures in more than 80% of patients. Ethosuximide does not suppress tonic-clonic seizures whereas valproate does.

Benign Partial Epilepsy with Centro Temporal Spikes

Benign childhood epilepsy with centrotemporal spikes (BECT) is the most common idiopathic partial epilepsy in childhood (10-20%) with excellent prognosis. [27] Age of onset is 3-13 years. Family history of febrile convulsion, epilepsy or subclinical centrotemporal sharp waves

may be seen in 40% of cases. Seizure semiology is characterized by brief hemifacial seizures becoming generalized, typically during sleep, or simple partial with unilateral paresthesias, tonic, clonic seizures, speech arrest, and inability to swallow with drooling. EEG shows diphasic and high voltage sharp wave centrotemporal activity. Drowsiness and slow sleep considerably increases the discharge rate. The sharp wave activity is unilateral in majority of patients and can be bilateral and also can be outside the centrotemporal area. Spontaneous remission with or without AEDs is the rule. BECT has been reported with brain lesions, which are not causally related. [28]

Benign Epilepsy with Occipital Paroxysms

Benign epilepsy with occipital paroxysms (BEOP) has its onset between 2-8 years in a child with normal development. [29] Seizures are characterized by visual phenomena like hemianopia, elementary or complex visual hallucinations or illusions followed by hemiclonic seizures or GTCS. Some patients may experience postictal diffuse headache, some with migraine like headache. EEG typically shows occipital spike-and-wave discharges commonly unilateral sometimes bilateral, synchronous or independent, disappearing during eye opening in the majority. sometimes associated with generalized or centrotemporal discharges. Similar EEG features are described with

symptomatic epilepsy due to occipital calcifications and gluten intolerance. [30] Outcome is favorable with AEDs.

Childhood Grand-mal Syndrome

Childhood grand mal syndrome (CGS) has its age of onset between 3-11 years with family history of epilepsy in 31% of cases and normal development. History of febrile seizures is common. Seizures are always generalized tonic clonic, sometimes associated with absence. In 50% EEG shows generalized spike-wave discharges with normal background. The outcome is favorable. Children presenting with grand mal seizures can have focal feature in 50% of EEGs. Hence, such cases are classified as partial going into generalized epilepsies.

Reflex Epilepsies – Photosensitive Epilepsy

The age of onset is 12-14 years, predominantly in females.48 Idiopathic GTCS is the commonest seizure but partial can be associated. Seizures can be precipitated by photic stimulation in symptomatic epilepsies also. Seizures may be spontaneous, photosensitive or self-induced. EEG shows generalized spike and wave discharges consistently to intermittent photic stimulation. Valproate is highly effective. Precipitating factors should be avoided.

Juvenile Absence Epilepsy

Juvenile absence epilepsy (JAE) occurs at puberty. Unlike the multiple clustered patterns in CAE, the absences in JAE are less frequent. Association with GTCS is frequent. Some patients may have myoclonic seizures. EEG features are similar to those observed in CAE. Polyspikes are often and discharge fragmentation may be seen. The drug of choice is valproate, which controls all seizures in 70% to 80% of patients. [32]

Epilepsy with Grand Mal on Awakening

Epilepsy with Grand Mal on Awakening occurs mostly in the second decade of life. The GTCS occur exclusively or predominantly shortly after awakening within first 2 hours regardless of time. Absence or myoclonic seizures are commonly associated and are precipitated by lack of sleep or overexertion. EEG shows generalized spike-and-wave discharges with disorganized background activity. Prognosis is favorable with treatment. Sleep deprivation should be avoided.[33]

Juvenile Myoclonic Epilepsy

Juvenile myoclonic epilepsy (JME) generally occurs during pre-or post-puberty.[34] Seizures are bilateral, single or repetitive, arrhythmic, myoclonic jerks predominantly in the arms. Patients may experience sudden falls but usually remain conscious. JME is commonly associated with GTCS on awakening and about one third of patients may have

absence seizures. EEG shows generalized polyspike-and-wave complexes of 4 to 6 Hz. The gene locus for JME is in chromosome 6P.[35]Valproate is effective in 86-90% of patients and to be continued for life long.

Autosomal Dominant Nocturnal Frontal Lobe Epilepsy

Autosomal dominant nocturnal frontal lobe epilepsy, which has been described recently,[36] is characterized by clusters of brief nocturnal hyperkinetic behavior or tonic seizures. There may be history of aura and consciousness is usually preserved. Onset is in childhood. The condition is often misdiagnosed as sleep disorder. Interictal EEG and neuroimaging are normal. Ictal EEG confirms partial seizures with frontal lobe involvement. Gene localization on chromosome 20q has been reported in this syndrome.[37] Carbamazepine monotherapy is often effective.

Symptomatic or Cryptogenic Localization-Related and generalized Epilepsies

Symptomatic Localization-Related and generalized Epilepsies in childhood cause most problems on account of their diverse semiological, etiological, and evolutional aspects. In the child population, putative etiologies include prenatal and perinatal pathology, developmental and neuronal migrational disorders, neurocutaneous syndromes, infections of the central nervous system, vascular insults and tumors. Often children with symptomatic epilepsies will have significant developmental delay with associated handicaps and multiple seizure.

AIM &OBEJECTIVE

- To find distribution of epileptic syndrome and epilepsy in children less than 12 years.

- To find Clinico- Aetiological profile of epileptic syndromes and epilepsy.

REVIEW OF LITERATURE

Epidemiological studies from the developed part of the world have revealed that epilepsy occurs with a prevalence rate of ~5/1000.4.5 [38,39] Although the prevalence rate of epilepsy in developing countries is reported to be twice that of the developing world,[42,43] this may be an artifact related to misdiagnosis and inclusion of symptomatic seizures, febrile seizures and inactive epilepsy. Regional causal factors such as cerebral cysticercosis [44] and hot water epilepsy[45] might have influenced the prevalence north, central and south India, have shown prevalence rates per 1000 population of 2.5 for Kashmir, [46] 3.6 for Bombay Parsis, [47]4.4 for Bangalore, [48] and 4.9 for Kerala (unpublished data). ILAE International classification of epilepsy in epileptic syndromes proposed by ILAE[49] is unsuitable for epidemiological purposes.

Sridharan and Murthy[51] recently underlook a meta-analysis of the prevalence data obtained from 20 community-based studies on epilepsy in India. After correcting for heterogeneity related to inter-study variation, the overall prevalence rate per 1000 was 5.3 (95% confidence intervals 4.3-6.4). The prevalence rates for urban areas was 5.1 (3.5-6.7), for rural areas, 5.5 (4.05-6.9); for men, 5.9 (3.5.-7.5)

Incidence studies, in contrast to cross-sectional studies, are most difficult to perform because they require longitudinal surveillance for several years. They are time consuming, expensive and labor intensive.

Studies from developed countries compute an incidence rate of ~50/100,000/ year.4.5. [50]

The only incidence data on epilepsy available from our country from Yelandur, Bangalore, reported a rate of 49.3/100,000/year. [49]

PATTERN AND ETIOLOGY

In the epidemiologic studies from India, primary generalized seizures accounted for 45 to 86%, while partial seizures accounted for 45 to 86%, while partial seizures with or without generalization formed 11 to 55%.[66]

PREVALENCE AND INCIDENCE OF REFRACTORY EPILEPSY

Nearly 20% of patients with active epilepsy would be resistant to antiepileptic drug (AED) treatment. In Kerala (population 30 millions), nearly 30,000 patients suffer from medically refractory epilepsy. Of these, 600 patients reside in the city of Trivandrum (population 600,000). To this number, nearly 30 patients with refractory epilepsy are added every year. Of the total population of India (900 million), about 900,000 people suffer from medically refractory epilepsy.

EPILEPSIES AND EPILEPTIC SYNDROMES IN CHILDHOOD: INDIAN EXPERIENCE

The pediatric Epilepsy Center at Bai Jerbai Wadia Hospital for children conducted study in 10 years. Of 2111 patients registered 1742

children with epilepsy were analyzed using the syndromic classification proposed by ILAE in 1989.

Localization- related Epilepsies

In this series of patients below 15 years, localization-related epilepsies accounted for 54.7% of the total cohort, Idiopathic localization-related epileptic syndromes formed 3.9% of the total cohort and 7.2% of localization-related epilepsies. In the series reported by Murthy et al[52] idiopathic localization-related epileptic syndromes accounted for 1.8% of childhood epilepsies.

Symptomatic localization-related epilepsies formed 48.8% of all localization-related an other hospital based study of epilepsies using the syndromic classification from India, symptomatic localization-related epilepsies accounted for 63% of patients with localization related epilepsies.[52]

The etiological spectrum of symptomatic localization-related epilepsies in this series included prenatal and perinatal pathology, infections of the central nervous system. developmental and neuronal migration disorders pediatric syndromes associated with dysmorphic features. Cerebrovascular diseases, and mesial temporalsclerosis. Solitary cysticercus granuloma was the putative risk factor in 50 children. Similar high incidence of solitary cysticercus granuloma was

observed in other large series of different types of epilepsies from India.[52,54]

Cryptogenic localization-related epilepsy accounted for 43.9% of the total cohort. The boundary of cryptogenic localization-related epilepsies is somewhat arbitrary because it depends on the extent of the diagnostic evaluation.[55]

Generalized Epilepsies and Epileptic Syndromes

This series included 620(35.5%) children with generalized epilepsies and epileptic syndromes; 40.6% idiopathic generalized epilepsies, 25.6% cryptogenic or symptomatic, and 33.7% symptomatic generalized epilepsies. Among the idiopathic epilepsies childhood and juvenile absence epilepsies together accounted for 0.9% of the total cohort. A similar low incidence was reported in other series from India.[52] Whereas Viani et al[56] had reported an incidence of 10.4%. the incidence of JME and grand mal on awakening is understandably low as they occur in adolescence.

Cryptogenic or symptomatic generalized epileptic syndromes accounted for 9% of the total cohort and for 25.6% of generalized epilepsies and epileptic syndromes WS accounted for a majority of the cases, 92.4%. Of the 147 patients with WS,124 (84%) were

symptomatic. The leading cause of symptomatic WS was prenatal and perinatal pathology.

In this series LGS was the epileptic syndrome in 0.4% of patients with epilepsy and in the population-based study from Japan reported by Oka et al[53] WS accounted for 4.4%.

Epilepsies and Epileptic Syndromes Undetermined Whether Focal or Generalized

They had 10 children with CSWS. Morikawa et al[49] and Tassinari et al[22] have reported 31 and 29 cases respectively in their large series.

Epilepsies and Epileptic Syndromes

A recent hospital-based study from south India also illustrated the rare occurrence of some of the syndromes described in the ILAE syndromic classification.[22]

Of the 2531 cases

Epilepsies and Epileptic Syndromes	Number of cases (%)
1. Localization related epilepsies	1591(62.9)
2. Generalized epilepsies and syndromes	299(11.8)
3. Epilepsies and syndromes undetermined whether focal or generalized	503 (19.9)
4. Special Syndromes	138 (5.4)

ETIOLOGICAL SPECTRUM

Despite the lack of uniform criteria upon which to base attribution of etiology, the available information shows that the ratio of undetermined to symptomatic etiology remains fairly constant across studies of incidence in both developed[50] and developing countries.[51] However, the relative frequency of different etiologies may very according to geographic location.1-4[61,62]

Etiological spectrum in hospital-based studies –Pre and post CT scan era

Etiology	Joshi et al (1970)20 Total number studied =1000 Symptomatic (%) =218(21.8)	Murthy et al (1998)23 Total number studied =2531 Symptomaic (%)=1255(49.5) AcuteSymptomatic(%) =522(41.6) Remote Symptomatic (%)=733(58.4)
Head trauma	93 (43)	19 (15)
CNS infections	64 (29)	456 (36)
Birth trauma	20 (9)	99 (8)
Intracranial tumors	12 (6)	40 (3)
Vascular diseases	13 (6)	296 (24)
Others	16 (7)	345 (28)

Prenatal and perinatal Pathology

In developing countries perinatal brain damage would account for 13-14% of the causes of epilepsy observed in children[65]. In the community based studies from India, the incidence of cerebral palsy or mental retardation among those with epilepsy was found to be 4.4% of those surveyed by Das and Sanyal, [66] 18.2% in those by Barucha et al, [63]and in 22.9% by Koul et al. [64] In a hospital-based study from south India, static encephalopathy related to perinatal brain damage accounted for 9% of cases.

Infections of the Central Nervous System

Infections and parasitic diseases of the central nervous system are the reasons most commonly cited for the higher incidence of the seizures in developing countries. Acute infections of CNS are risk factors for epilepsy. In a retrospective cohort study from Rochester, Minnesota,[50] that followed people of all ages for the development of unprovoked seizures, CNS infections increased seizure risk 11-fold; the risk of seizures varies across different types of CNS infections. [67]

Bacterial meningitis

The risk of developing epilepsy is four-fold following bacterial meningitis, especially in patients with acute symptomatic seizures. [67] Makrs et al [69] found a significant association between bacterial meningitis in early childhood (before the age of 4) and intractable epilepsy due to mesial temporal sclerosis. The cerebral injury that has the highest association with the subsequent development of unprovoked seizures is cerebral ischemia. [70,71]

Neurotuberculosis

Epilepsy occurs in~ 10% of cases with neurotuberculosis and almost always occurs with signs of focal central nervous system involvement. [73,74] Though incidence of tuberculous meningitis is still high in India, [72] there are no prospective long-term cohort studies to determine the incidence of epilepsy. In one study tuberculous meningitis was the putative factor in 3.5% of patients with remote symptomatic epilepsy. [62]

Japanese encephalitis

Epilepsy is a rare sequelae and only one of 75 patients followed for five years had epilepsy. [75]

Neurocysticercosis

Like in the other developing countries, neurocysticercosis is a growing problem in India and is increasingly recognized as the cause of epilepsy. [76,77] Before the availability of CT, the frequency of neurocyscticercosis as a cause of epilepsy in India was reported to vary from 2.2% to 9.6%.[78,79] After the availability of CT/MR imaging, neurocysticercosis has been found to be the cause in 9 to 423/2531.9[80]Post-mortem and other studies suggest that asymptomatic cysticercosis is also common. [81]

Solitary cysticercus granuloma

In India single enhancing lesion (Fig.4.3) is a common finding in CT brain scans of patients with epilepsy and has been reported from all the provinces. [82] Biopsy results of these lesions have indicated varied pathology; cysticercus, tuberculomas, pyogenic abscess, meningencephalitis, and metastatic secondaries. However, in countries where neurocysticercosis is endemic, small CT enhancing lesion more often represents a solitary cysticercus granuloma (SCG). [83,84] Most of the studies on the frequency of SCG in India are culled from neurologic settings. In patients with partial seizures this lesion accounted for 26% of etiological factors in two different studies.23, [85,86] Of the 406 cases of neruocysticercosis described by Singhal and Ladiwals56 88.6% had

SCG and in our series of 432 cases of neurocysticercosis, SCG accounted for 62%. Rajashekar and Chandy63 validated some diagnostic clinical and CT criteria for “solitary cerebral cysticercus granuloma” in patients with seizures. The clinical criteria are : seizures as the initial symptom, with no evidence of persistent intracranial pressure, no progressive neurologic deficits, and no active systemic disease. The CT diagnostic criteria are evidence of a solidary contrast enhancing lesion measuring less than 20mm, without shift of the midline structures due to surrounding edema.

Single small cerebral calcific CT lesion

A single small cerebral calcific lesion measuring less than 20 mm is one of the findings seen in CT brain scans of patients with epilepsy in geographical areas where neurocysticercosis is endemic. [84] [88,90] In a hospital-based study this lesion was the putative risk factor in 9% of patients with localization-related epilepsy. [90] This CT lesion is evidence of cysticercal brain parenchymal involvement previous to the calcification becoming evident in CT, particularly in those geographic regions where human cysticercosis is endemic. [59,60,87,90]

Reflex Epilepsies

Epilepsies with seizures precipitated by specific modes of activation are designated reflex epilepsy. In Europe, an evoking factor had been found in 5-6.5% of patients with epilepsy.[99] Lower frequencies

2.6-2.8% have been reported from India. [100,101] The pattern of reflex epilepsies is considerably different in the tropics. Eating epilepsy[100-105] and self-induced epilepsy [100,107,108] are the common reflex epilepsies reported form India. Photosensitive epilepsy is by far the most common from of reflex epilepsy reported from developed countries. The reported frequency of photosensitive epilepsy among patients with epilepsy from India varied between 0.6% to 3.5% [108,109]

Out come of Epilepsy :

Once recurrent epileptic seizures have occurred, an important issue is the probability of early remission. In a large prospective study 57% of children, 2 years after a diagnosis of epilepsy, had a 'good outcome'(a remission which had lasted more than 12 months).12% had a 'fair outcome'(a remission which had lasted 6-12 months) and 31% had a 'poor outcome'(a remission which had lasted less than 6 months). [110] Another study reported that at 2 years after diagnosis of epilepsy 53% of children had a 'good outcome' (a remission of more than a year) 8% had a 'bad outcome' (two or more antiepileptic drug failures and at least one seizure per month over an 18 month period) and 38% had an 'intermediate outcome'. [111] In about 80% of children who were followed for 4 or more years the outcome was similar to that at 2 years. However, around half of those with an intermediate outcome at 2 years achieved remission and 8% became intractable. Factors which are

associated with the likelihood of a poor outcome include young age at seizure onset, symptomatic etiology, an initial presentation with status epilepticus or infantile spasms and high initial seizure number. [112,113,114]

METHODOLOGY

Observational study by analyzing medical records, history and physical examination of patients and analyzing EEG & Neuro imaging.

STUDY DESIGN

Cross sectional & longitudinal study. (Cross sectional-already diagnosed attending epilepsy clinic)

(Longitudinal –patients added to the epilepsy registry every month, during the course of the study September 2004-December 2005. And follow up of epileptic population to establish clinical and prognostic profile.)

STUDY POPULATION

Patients Attending Seizure disorder clinic.

DURATION OF THE STUDY

December 2003- December 2005.

MATERIALS

Epilepsy registry and medical records obtained from patients.

INCLUSION CRITERIA

a. Generalised Seizures

b.Localised seizures

a. Unequivocal generalized (or) localized disorder.

b. Seizures following Post Meningitic&encephalitic sequale.

EXCLUSION CRITERIA

a. Hysterical seizures

b. Seizure like disorders. (Paroxysmal disorders)

c. Neonatal seizures.

d. Febrile seizure

DEFINITION OF VARIABLES

Epileptic syndrome is defined five axial diagnostic scheme depending upon.

1. Behavioural phenomenology
2. Diagnostic seizure type.
3. Generalised/Localised
4. Aetiology
5. Functional disability

METHODS OF DETERMINATION

- CT
- EEG
- Clinical Examination
- Follow up

OUTCOME

1. To find distribution of epileptic syndromes and epilepsies.
2. To find commonest etiological factors attributed to epileptic syndrome and epilepsy
3. To establish pattern of clinical semiology of seizures in individual type
4. To find out outcome of epilepsy and factors related to outcome.

PROPOSED ANALYSIS

ETHICS

OBSERVATION & RESULTS

EPILEPSY AND EPILEPTIC SYNDROME PROFILE

DISTRIBUTION (TOTAL:200 CASES)

GENERALIZED EPILEPSY (108 CASES)

TONIC CLONIC	82
TONIC	8
MYOCLONIC	15
ABSENCE	2
ATONIC	1

MALE	-	75CASES
FEMALE	-	33CASES

MEAN AGE OFONSET -1 ½ YEARS

PARTIAL EPILEPSY (92CASES)

SIMPLE PARITAL	4
COMPLEX PARTIAL	76
With secondary GENERALISATION	12

MEAN AGE OFONSET - 4YEARS

MALE	-	60CASES
FEMALE	-	32CASES

UNCLASSIFIED(0CASES)

EPILEPTIC SYNDROMES OBSERVED(16CASES)

WEST SYNDROME	2
LENNOXGESTAUT SYNDROME	4
CHILDHOOD ABSENCE	2
BENIGN PARTIAL EPILEPSY OF CHILDHOOD	6
INFANTILE EPILEPTIC ENCEPHALOPATHY	1
NOCTURNAL FRONTAL LOBE EPILEPSY	1

ETIOLOGY

GENERALISED EPILEPSY(108CASES)

Asphyxia	42
Neuronal	7
Infectious sequale	11
TORCH	2
Idiopathic(reflex epilepsy-1)	41
Infantile epileptic encephalopathy	1
SCTCEL	1
Specific syndromes (sturge Weber)	1
Tuberculoma	1

Neurocysticercosis	1

LOCALIZATION RELATED EPILEPSY(92CASES)

ETIOLOGY

Tuberculoma	26
Neurocysticercosis	12
Neuronal migration disorder	3
Asphyxia	19
Post infectious sequale	7
Calcified granuloma	3
Cerebro vaslular Accident	1
Tuberous sclerosis	1
Nocturnal frontal lobe epilepsy	1
Idiopathic(Including BPEC-6)	19

EEG findings

Generalised epilepsy

Cerebral Dysfunction	6
Sharp waves and spike waves	46
Sharp waves	29
Hypsarrhythmia	1
Sharp waves spikes C 3HZ Freq	1
Polyspikes	3

Back gorund slowing with Sharpwaves and spikes	8
Normal	10

Localiastion related epilepsy

Focal disharges	18
Diffuse discharges	66
Normal	8

OUTCOME OF EPILEPSY

(followed upto 2 year-129cases(71%))

< 2year follow up-71cases(29%))

Good out come	50
Intermediate out come	50
Bad out come	29

Factors Associated with Bad Out come(total 26 cases)

1) Young Age of onset<1year(10 out of 26cases)
2) Symptomatic Etiology -(29 out of 26cases)
3) Intial Presentation (14 out26cases)
   With Status epilepticus
4) Infantile spasms -(3 out of 26 cases)
5) Initial high number of seizures-(8 out of 26 cases)

DISCUSSION

In India and other countries, studies on epilepsy are mostly community based. there are only few about hospital based studies.

In our study generalization related epilepsy is the most common category compared with other studies in India like rajyadyaksha, shah etal[115] and J.M.K. Murthy, Yangala etal,[52] where localization related epilepsy is the commonest category.

In our study, we used ILAE 1981 classification of epileptic seizure types rather than 1989 classification of epilepsy and epileptic syndromes. we used 1989 classification for epileptic syndrome only.

As the main objective of our study is founding of individual known etiological aspects like perinatal events CNS infections, cortical dysplasia etc., we are not using 1989 classification for epilepsy as done in other Indian studies.

But by observing clinical data, EEG, CT findings cryptogenic epilepsy and epileptic syndromes account for 7.5% of total cases (15 cases) WS-1, LGS-1 Other cryptogenic localisation related epilepsies-13 which is low as compared with other Indian studies where cryptogenic epilepsies account for 34% rajadyaksha shah etal[115] and 41.7% in murthy etal. [52]

In our results we included the cryptogenic variety of localization related epilepsy in idiopathic category to avoid confusion between known etiology and presumed etiology.

Regarding Idiopathic Epilepsy our study accounts for 30% of total cohort which is similar with rajadyaksha et al[115] (30%) but high as compared with Murthy et al (7.1%)[52]

Regarding symptomatic Epilepsy, our study group accounted for 70% of cases which is higher than other Indian studies is 36% in Rajadyaksha et al[115] and 42% in Murthy et al. [52]

Regarding symptomatic Epilepsies, perinatal Asphyxia is the single most common (37.7% of generalized Epilepsy) Etiology found in generalized Epilepsy followed by infections segued as reported in other studies in India and neighboring countries[116,117] and Rochester study. [50]

SCTEL accounts for major cause (47%) of localization Related epilepsy as mentioned in other studies. Regarding unclassified seizures and epilepsy of undetermined origin, none of tem reported as compared with 19.9% in Murthy et al[52] and 9.6% in Rajadyaksha et al. [115]

Epileptic syndromes accounted for 8% of total cases in our study as compared with 16.3% in Rajadyaksha et al[115] and 11.2% in Murthy et al. [52] probably reflects advanced technologies like video EEG and Advanced Neuro imaging in the above mentioned study centres.

Among Epileptic Syndromes Benign Epilepsy with centero temporal spikes is the commonest entity followed by LGS as similar with Rajadyaksha etal, [115] but JME is the commonest entity in Murthy et al[52] as this study included <15yrs age group, upper age limit of this study will obviously include JME since its occurrence mainly in the adolescence period.

In SCTEL Responsible for major cause of symptomatic localization related Epilepsy, Tuberculoma is the commonest Etiology as compared with other studies mainly Rajadyaksha etal[115] and Murthy et al[52]which reported Neurocysticerosis as a major cause.

Regarding EEG findings spikes and sharp waves is the commonest category observed both in generalized and localization Related Epilepsy. But focal discharges only observed in 18% as partial Epilepsies, since most of EEG done in interictal period Ictal EEG records less commonly practiced in our hospital.

Regarding outcome of Epilepsy as described by Berg and shiner et al, [111] In 129 cases who are regularly followed up more than 1 year, 29 cases(22%) has Bad out come, that will cause intractable Epilepsy in the future. factors responsible for bad outcome observed in our study are

1) Young Age of onset

2) Symptomatic Etiology

3) Initial presentation with status Epileptics

4) Infantile spasms

5) High number of Initial Seizures

Of these, symptomatic Etiology and Initial presentation with status Epileptics are the two important risk factors associated with majority of cases (>60%)

CONCLUSION

1) **Generalised epilepsy is the most common category.**

   - ❖ Accounts for 54% epilepsy (total 108 cases)
   - ❖ Among this **tonic clonic** convulsions is the most common clinical entity followed by myoclonic seizures.
   - ❖ **Perinatal Asphyxia** is the single most common etiology followed by infectious Sequale.

2) **Localisation related epilepsy**

   Is the second most common category-92 cases (46%)

   - ❖ Among this Category- **Complex partial** seizure is the commonest clinical entity.
   - ❖ **SCTEL(Including Tuberculoma, Neurocysticercosis, Calcified Granuloma)**

   Is the Most common etiology observed in localization related epilepsy followed by perinatal asphyxia.

3) **In unclassified Seizures none of them observed**

4) **Epiletic Syndromes(16cases)**

   - ❖ Account for (8%) of total cases
   - ❖ Among this **BPEC** is the most common epileptic syndrome followed by LGS.

- As JME(which is the commonest epileptic Syndrome in India) usually occurs in Adolescence none of the cases reported

5) Among EEG findings

Sharp waves and spike waves is the most common entity in Generalised epilepsy

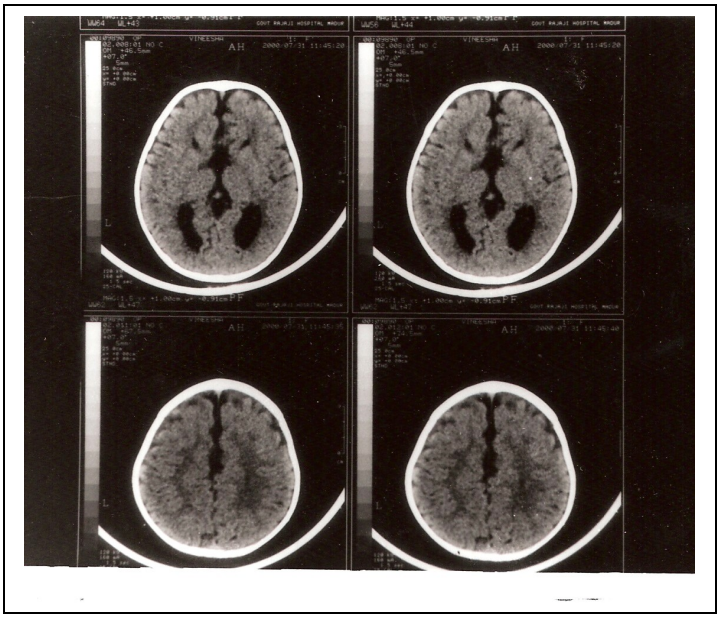
Focal EEG Discharges found only in 18 cases (9.8%) of localization related epilepsy

6) Regarding out come of epilepsy, who had more than 1 year Follow up (total 129cases), 29 cases (22%) Belong to Bad Out Come, will go for intractrable epilepsy.

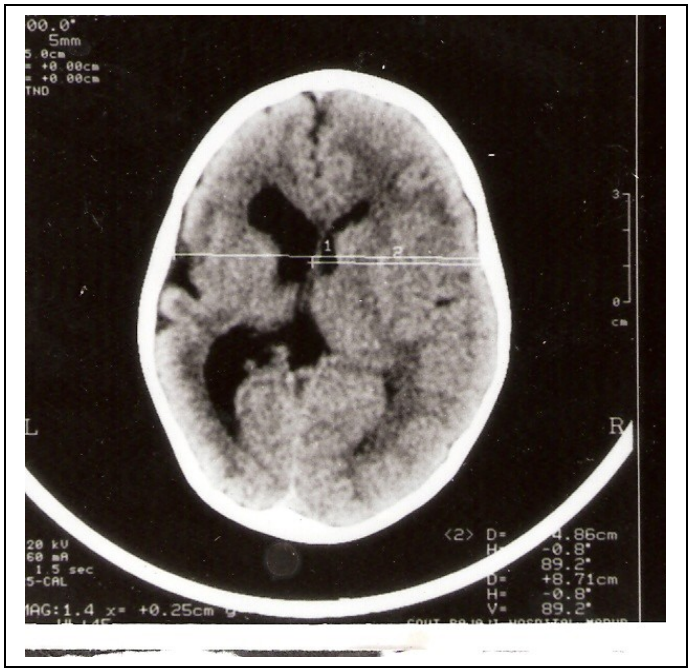
Factor Associated with Bad outcome Observed in Our study Are

1) Young Age at onset (7 cases)
2) Sympotomatic etiology (26cases)
3) Initial Presentation (14cases)
with Status Epilepticus
4) Infantile spasms- (3cases)
5) Initial High Number of Seizures- (5cases)

AGENESIS OF CORPUS COLLOSUM

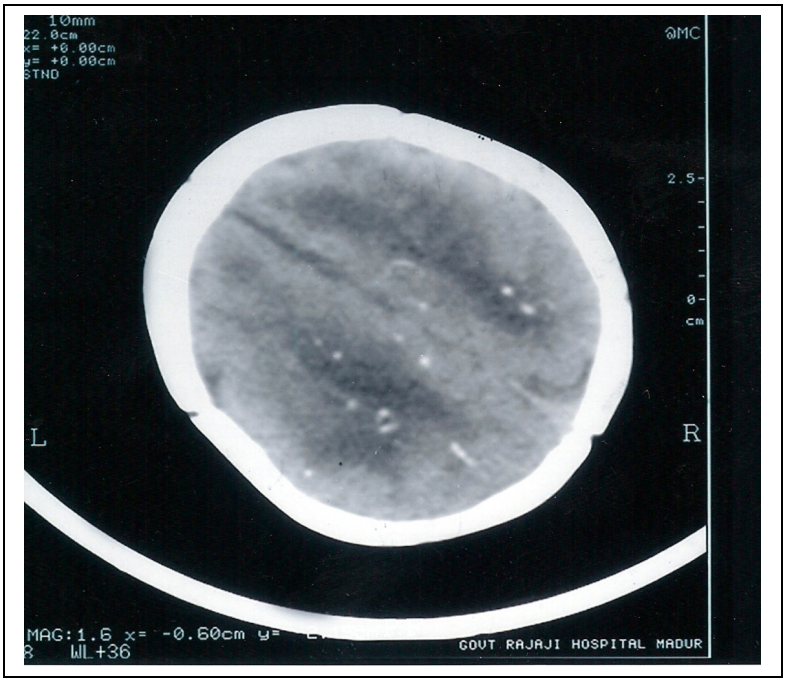


CORTICAL HEMI ATROPHY

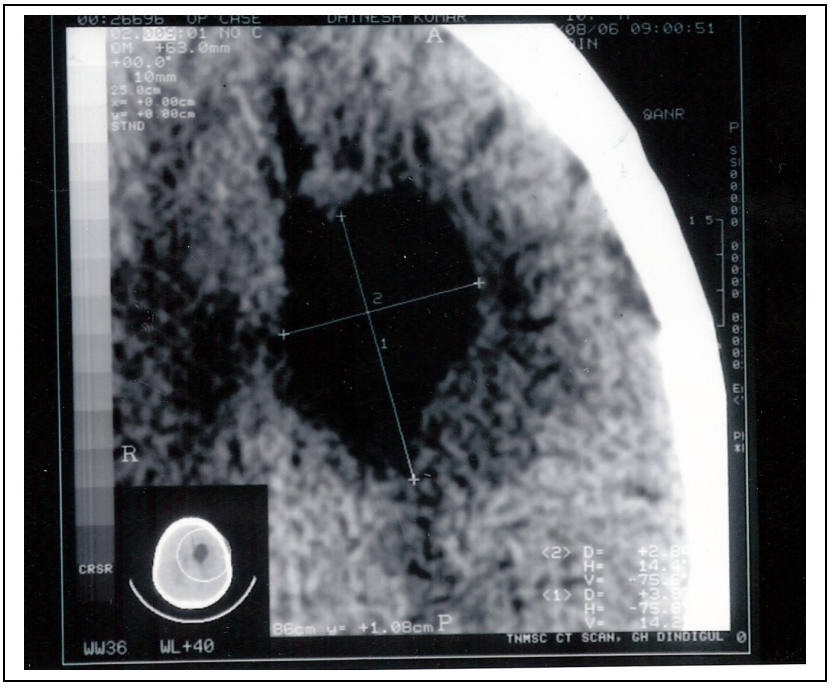


BASAL GANGLIA CALCIFICATION

PERIVENTRICULAR CALCIFICATION

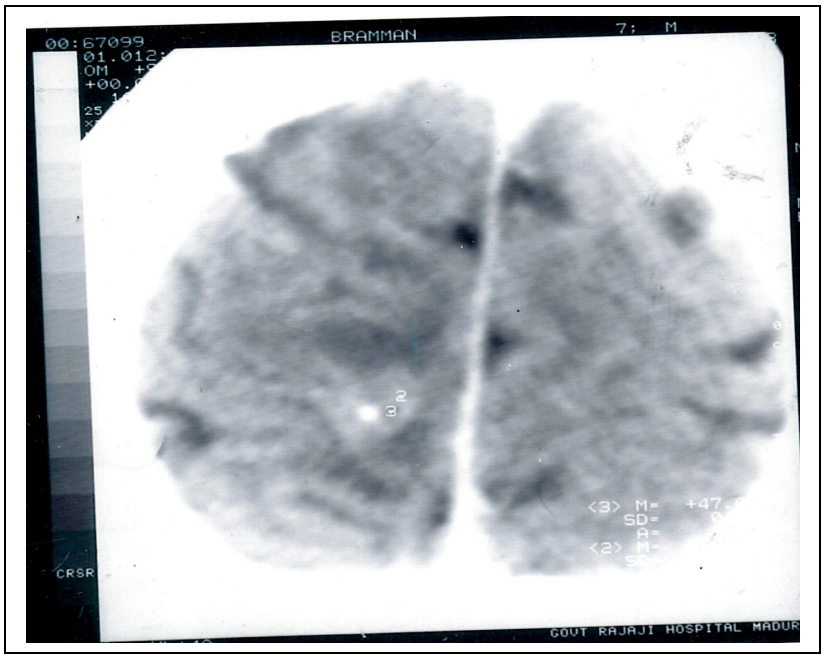


NEURONAL MIGRATION DISORDER-PORENCEPHALIC CYST



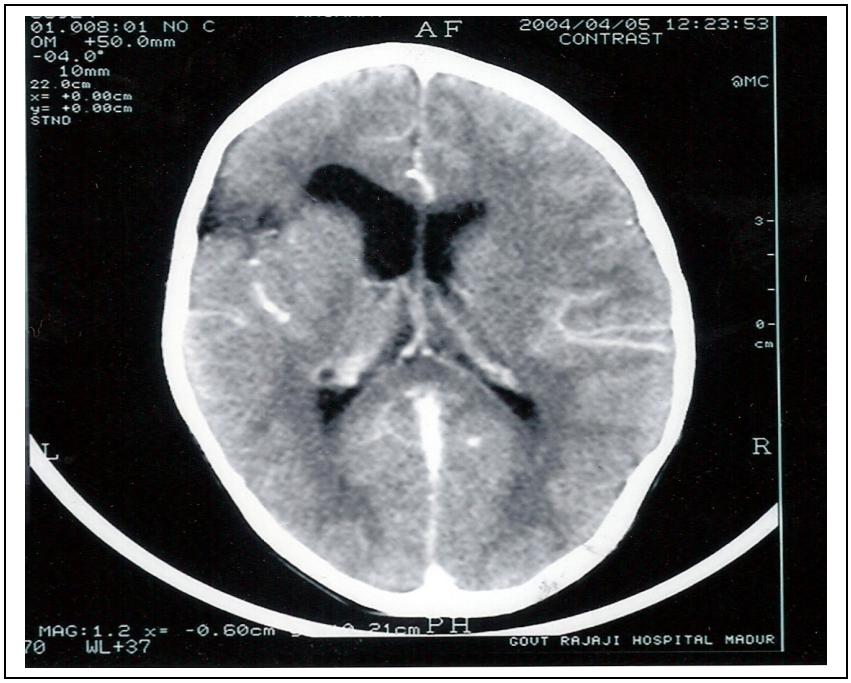
CORTICAL INFARCT WITH GLIOMATOUS CHANGE

NEUROCYSTICERCOSIS WITH DOT SIGN

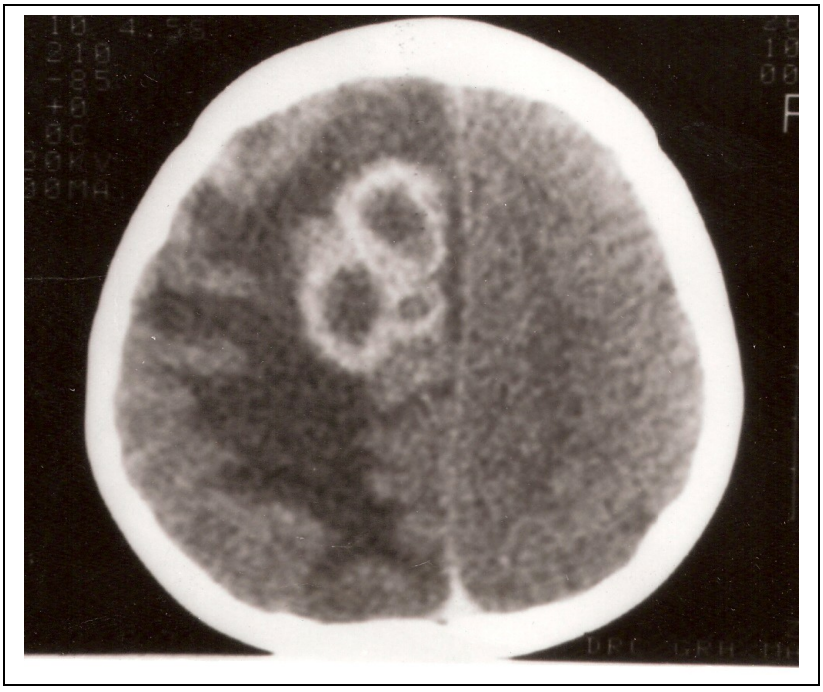


TUBERCULOMA WITH PERILESIONAL OEDEMA

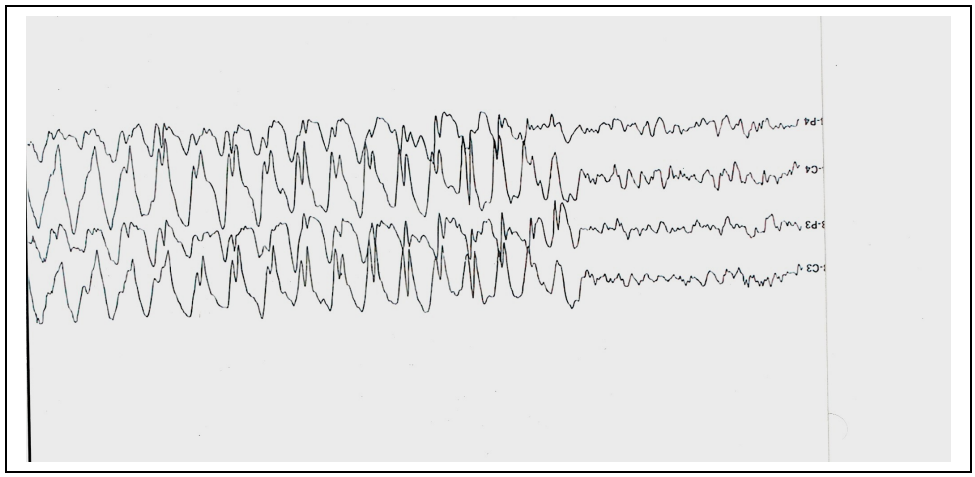
MENINGITIC SEQUALE WITH VASCULITIC INFARCT



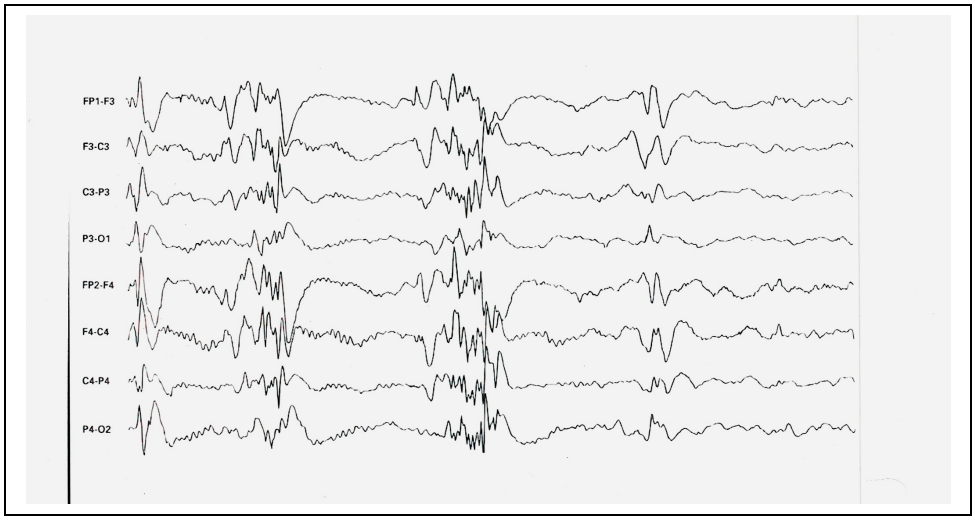
MULTIPLE TUBERCULOMA



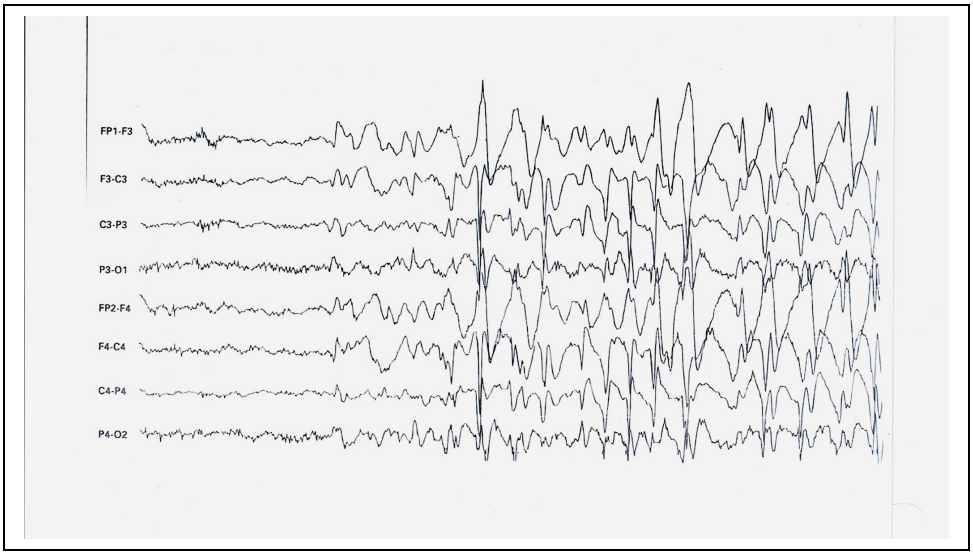
SPIKES ONLY



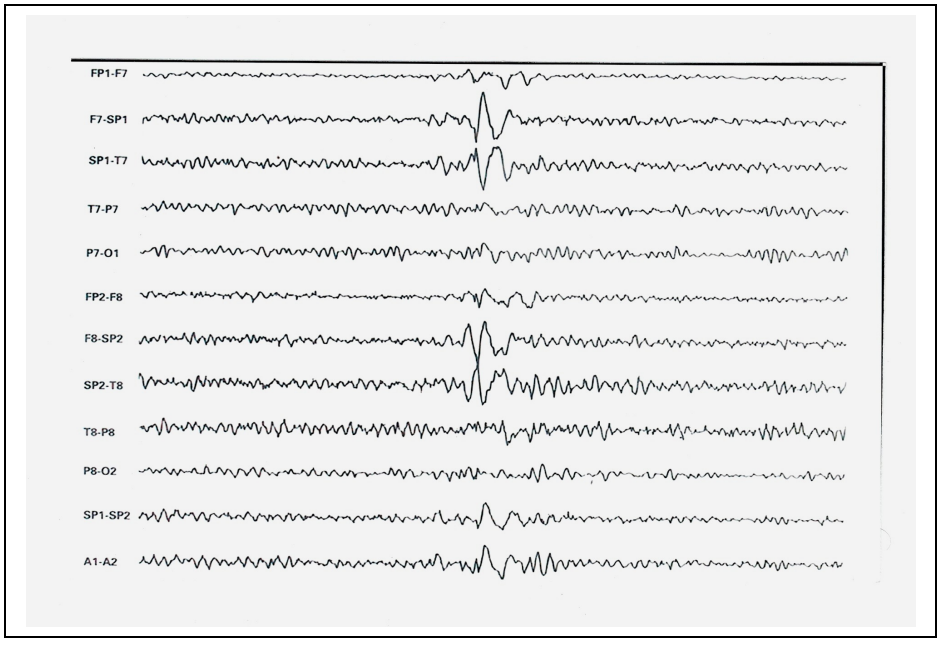
POLYSPIKES-LENNOX GESTAUT SYNDROME



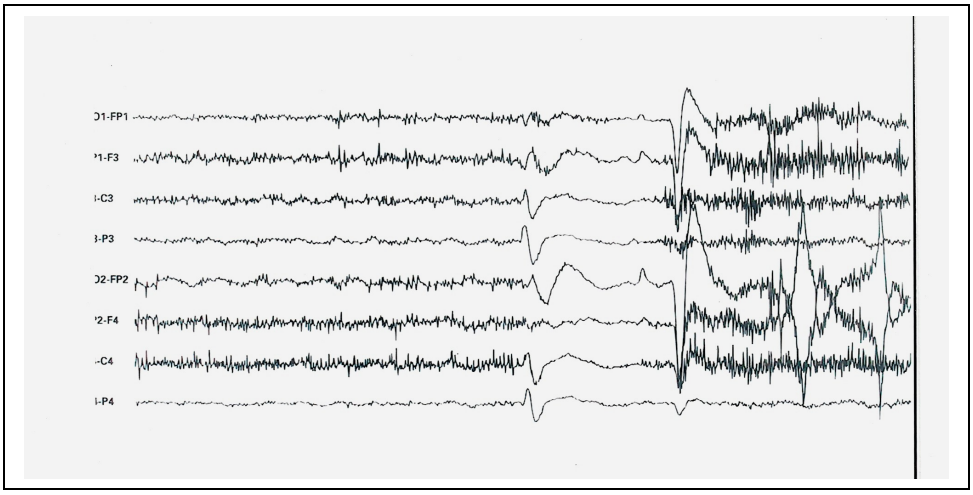
SPIKES AND SHARPWAVES



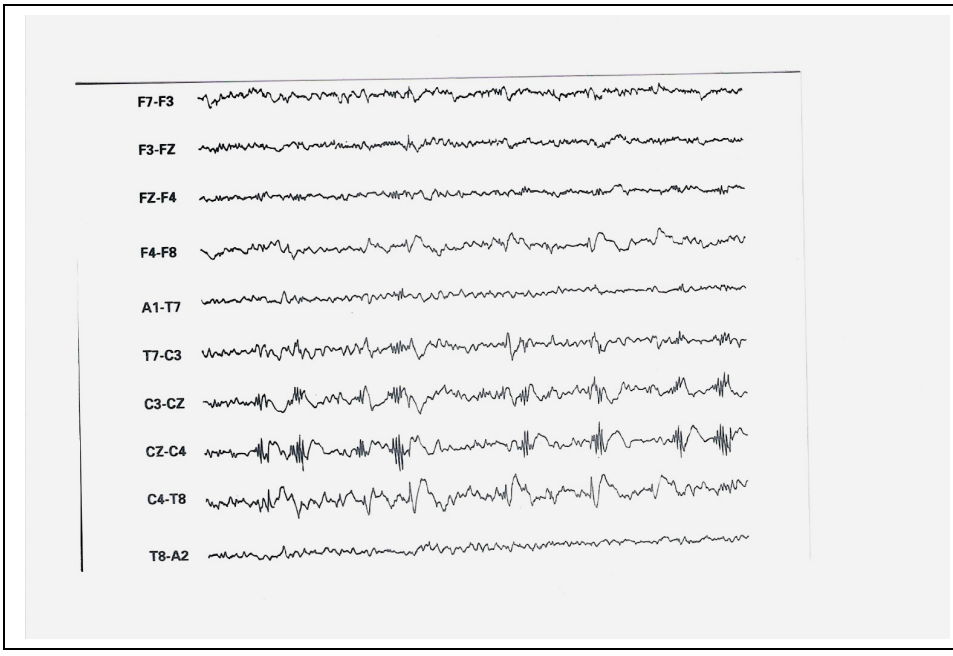
SHARP WAVES ONLY



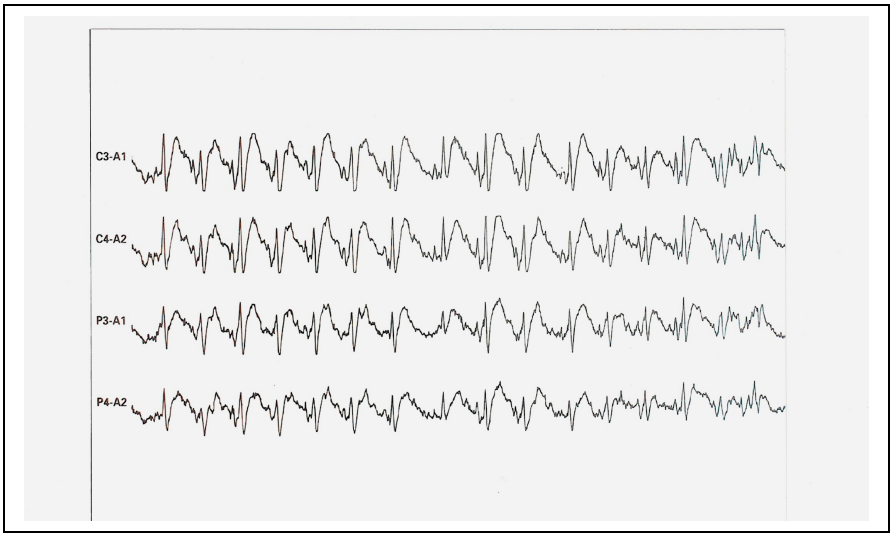
LEFT FOCAL DISCHARGE



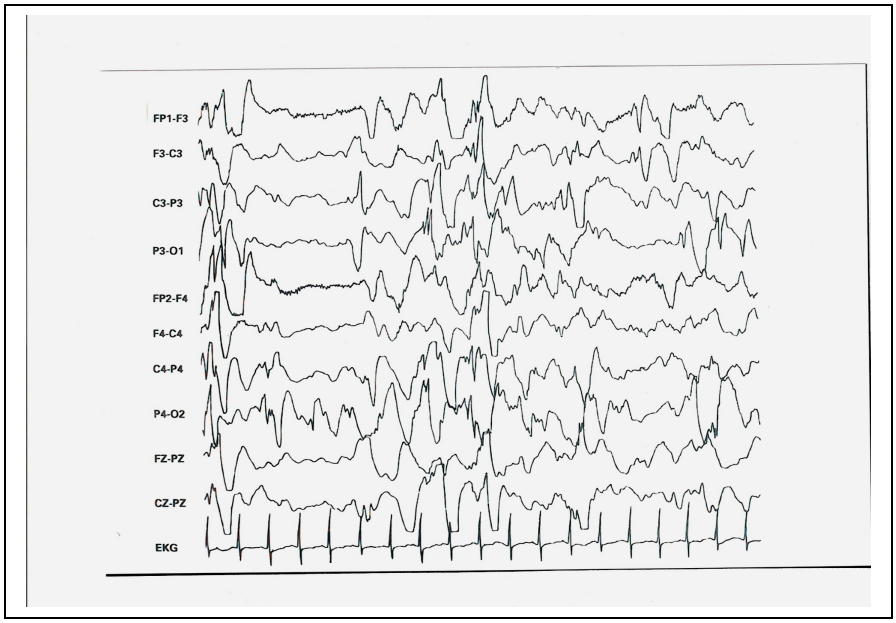
CENTERO TEMPORAL SPIKES



ABSENCE SEIZURES-3 HZ SPIKES



HYPSARRHYTHMIC PATTERN-WEST SYNDROME



EPILEPTIC SYNDROMES - PROFORMA

Name : Informat :

Age : Reliability :

Sex : Address :

PRESENTING COMPLAINTS :

Hallucinations

Aura

Convulsions - Generalised

- Localised
- Unequivocal generalized or localized
- Autonomic
- Psychomotor

Time of seizure - Day / Night / Awakening

Duration

Loss of consciousness / Impairment of consciouness

Involuntary micturtion

Tongue biting

Automatisms

Frequency of seizure (clusters / episodes)

Behavioural changes (Crying, laughing)

Provoking factors

Association with fever

Time of onset of seizure with fever

Association with lunar cycle

PAST H/O

Previous seizures - Type

- Age of onset
- Recurrence per year

Previous head injury

ANTENATAL	-	Exanthematous fever
	-	Drug intake
NATAL	-	Birth asphyxia +
POST NATAL	-	Jaundice
	-	Seizures
DEVELOPMENTAL H/O	-	Delayed / Normal
FAMILY H/O	-	Consanguinous / Non consanguinous
	-	Seizures
IMMUNIZATION H/O	-	BCG Scar +/-
CONTACT H/O	-	+ / -
SOCIAL H/O	-	Unhygienic food practices
	-	Veg / Non Veg
	-	Sanitation
	-	Animal rearing practices

GENERAL EXAMINATION

- Congenital Anomalies
- Neuro cutaneous markers
- Scars due to fall

HC WT HT

CNS EXAMINATION

- Higher functions Delayed / Retarted / Normal
  - Language
  - Speech
  - Communication

* Mental retardation + / -
* Motor system
* Sensory system

* Reflexes
* Gait
* ANS

OTHER SYSTEMS

INVESTIGATIONS

- EEG
- CT SCAN
- MRI
- URINE METABOLIC SCREENING
- TORCH PROFILE

ANY RECOGNIZED CLINICAL SYNDROME

DIAGNOSIS - Classification according to ILAE :

TREATMENT

FOLLOW UP Remissions & Recurrrence

BIBLIOGRAPHY

1. Gastaut H. Clinical an delectroencephalographic classification of epileptic seizures Epilepsia 1970;11:102-13.
2. Commission on classification and Terminalogy of the international League Against Epilepsy : Proposal for revised clinical and electroencephalographic classification of epileptic seizures, Epilesia 1981;22:489-501.
3. Aicardi J. Classification of epileptic seizures and epilepsies. In: Aicardi J,ed. Epilepsy in children. New York, Raven Press, 1994 2nd Edition;9-17.
4. Luders HO, Burgess R, Naochtar S. Expanding the International Classification of Seizures to provide localization information. Neurology 1993;43:1650-4.
5. Roger J, Bureau M, Dravet CH, Dreifuss FE, Perret A, Wolf P. Epileptic syndromes in infancy, childhood and adolescence. London John Libbey, 2nd Edition, 1992.
6. Aicardi J. Epilepsy in children. New York Raven Press, 2nd Edition,1994.
7. Haslam RA, Febrile seizures, In Nelson WE, Behrman RE,Kilegman RM. Arvin, AM. eds. Textbook of Pediatrics Prism Book Pvt Ltd. Bangalore 1996; 15th Edition, 1691.

8. Clarke M,Gill J, Noronha M, Mckinlay I, Early infantile epileptic encephalopathy with suppression burst: Ohtahara syndrome. Dev Med Child Neurol 1987;29:520-8.
9. Harding BN, Boyd SG. Inractrable seizures from infancy can be associated with dentate –olivary dysplasia. J Neurol Scie 1991;104:157-65.
10. Pedespan JM, Loiseau H, Vital A, Marchal C, Fontan D, Rougier A. Surgical treatment of an early epileptic encephalopathy with suppression bursts and focal cortical dysplasia. Epilepsia 1995;36:37-40.
11. Jeavonds PM, Livet MO. West syndrome: Infantile spasms. In: Roger J. Bureau M, Dravet CH. Drifuss FE, Perret A,Wolf P, eds. Epileptic syndromes in infancy, Childhood and Adolescence. London, John Libbey, 2nd Edition, 1992:53-65.
12. Bednareck N, Motte J, Soufflet C, Plouin P, Dulac O. Evidence for late onset infantile spasms. Epilepsia 1998;39:55-60.
13. Chiron C, Dulac O, Beaumont D, Palacios L, Pajot N, Mumford J, Therapeutic trial of vigabatrin in refractory infantile spasms. J Child Neurol 1991;6 (Suppl 2):552-9.
14. Chugani HT, Shields WD, Shewmon DA, Olson DM, Phelps ME, Peacock WJ. Infantile spasms : PET identifies focal cortical

dysgenesis in cryptogenic cases for surgical Treatment. Ann Neurol 1990;27:406-13.

15. Dravet C, Bureau M, Gurrini R, Giraud N, Roger J. Severe myoclonic epilepsy in infants, In: Roger J, Bureau M, Dravet CH, Dreifuss FE, Perret A, Wolf P, eds. Epileptic syndromes in infancy, childhood and adolescence, London, John Libbey, 2nd Edition 1992:75-88.
16. Dravet C, Bureau M, Roger J. Benign myoclonic epilepsy in infants. In: Roger J, Bureau M, Dravet CH, Dreifuss FE, Perret A, Wolf P,eds. Epileptic Syndromes in Infancy. Childhood and Adolescence. London, John Libby, 2nd Edition, 1992:67-74.
17. Tassinari CA, Bureau M, Thomas P. Epilepsy with myoclonic absences. In: Roger J, Bureau M, Dravet CH, Dreifuss FE, Perret A, Wolf P.eds Epileptic Syndromes in Infancy, Childhood and Adolescence. London, John Libby, 2nd Edition, 1992:151-60.
18. Dulac O, Guyen TN. The Lennox-Gastaut syndrome. Epilepsia, 1993; 34 (Suppl.7): S7-S17.
19. Dichter MA, Brodie MJ. New antiepileptic drugs. N Engl J Med 1996;334:1583-90.
20. Andermann F, Oliver A, Gotman J, Sergent J, Callosotomy for the treatment of patients with intractable epilepsy and the

Lennox-Gastaut syndrome. In: Niedermeyer E, Degan R, eds. The Lennox-Gastaut syndrome. New York , Alan R Liss, 1988:361-79.

21. Hoeppner JA, Grote CL, Morrell F, Whistler W, Brocken C, Long term follow-up of cognitive and behavioral function after surgery for Landau Kleffner syndrome. Epilepsia 1993;34(Suppl 6):72..

22. Tassinari CA, Bureau M, Dravet C, Dalla Bernardina B, Roger J. Epilepsy with continuous spikes and waves during slow sleep. In: Roger J, Dravet C, Bureau M, Dreifuss FE, Perret A, Wolf P, eds. Epileptic syndromes in infancy, childhood and adolescence, London, John Libbey, 2nd Edition. 1992:245-563

23. Bercovic SF, Andermann F, Melanson D, Ethier RE, Feindel W, Gloor P. Hypothalamic hamartomas and ictal laughter: evolution of a characteristic epileptic syndrome and diagnostic value of magnetic resonance imaging. Ann Neurol 1988;23:429-39.

24. Mohio S, Morioka T, Fukui M,Goto Y. Surgical treatment of intractable seizures due to hypothalamic hamartoma. Epilepsia 1994;35:514-9.

25. Loiseau P.Childhood absence epilepsy. In: Roger J, Bureau M, Dravet CH, Dreifuss FE, Perret A, Wolf P, eds. Epileptic

syndromes in Infancy, childhood and adolescence, London, John Libbey ; 2nd Edition, 1992:135-50.

26. Penry JK, Porter RJ, Dreifuss FE. Simultaneous recording of absence seizures with video tape and electroencephalography. A study of 374 seizures in 48 patients. Brain 1973;98:427-40.

27. Lerman P.Benign Partial epilepsy with centro-temporal spikes. In Roger J, Bureau M, Dravet CH, Dreifuss FE, Perret A, Wolf P, eds. Epileptic syndromes in infancy, childhood and adolescence. 2nd Edition, Londoan, John Libbey, 1992:189-200.

28. Morikawa T, Osawa T, Ishihara O, Seino M. A reappraisal of “benign epilepsy of children with centro-temporal EEF foci”. Brain Dev 1979;1:253-65.

29. Gastaut H. Benign epilepsy of childhood with occipital paroxysms. In: Roger J, Bureau M, Dravet CH, Dreifuss FE, Perret A, Wolf P, eds. Epileptic Syndromes in Infancy, Childhood and Adolescence. London, John Libbey, 2nd Edition. 1992:201-17.

30. Magaudda A, Dalla-Bernardina B, De Marco P, Sfaello Z, Longo M. Bilateral occipital calcification, epilepsy and celiac disease: Clinical and neuroimaging features of a new syndrome. J Neuro Neurosurg Psychiatry 1993;56:885-9.

31. Binnie CD, Jeavons PM. Photosensitive epilepsies. In: Roger J, Bureau M, Dravet CH, Dreyfuss FE, Perret A, WolfP, eds.

Epileptic syndromes in infancy, childhood and adolescence. 2nd Edition, London,. John Libbey, 1992:299-305.

32. Wolf P.Juvenile absence epilepsy. In: Roger J, Bureau M, Dravet CH, Drifuss FE, Perrel A, Wolf P., eds. Epileptic syndromes in infancy, childhood and adolescence. 2nd Edition, London, John Libbey 1992:307-12.
33. Wolf P.Epilepsy with grand mal on awakening. In Roger J, Bureau M, Dravet CH, Dreyfuss FE,Perret A. Wolf P, eds. Epileptic syndromes in infancy childhood and adolescence. London,. John Libbey, 2nd Edition, 1992:329-41.
34. Wolf P.Juvenile myoclonic Epilepsy, In Roger J, Bureau M, Dravet CH, Dreifuss FE, Perret A, Wolf P, eds. Epileptic syndromes in infancy, childhood and adolescence. London, John Libbey, 2nd Edition, 1992:313-27.
35. Weissbecker KA, Durner M. Janz D, Scarmelli A, Spence MA. Confirmation of linkage between juvenile myoclonic epilepsy and HLA region on chromosome 6. AM J Med Genet 1991;38:32-6.
36. Scheffer I, Bhatia K. Lopes Cendes I et al: Autosomal dominant nocturnal frontal lobe epilepsy: A distinctive clinical disorder. Brain 1995; 118:61-73.
37. Philips HA, Schaffer IE, Bercovic SF, Hollway GE, Sutherland GR, Mulley JC. Localization of a gene for autosomal dominant

nocturnal frontal lobe epilepsy to chromosome 2oQ13.2. Nature Genet 1995;10:117-8.

38. Hauser WA, Kurland LT. The epidemiology of epilepsy in Rochester,Minnesota, 1935-1967. Epilepsia 1975;16:1-66.

39. Sander JWAS, Shorven SD. Epidemiology of the epilepsies, J Neurol Neurosurg Psychiatry 1996; 61:433-43.

40. Sander JWAS. Some aspect of prognosis in the epilepsies : A review.Epilepsia 1993;34: 1007-16.

41. Shinnar S.Berg AT.Does antiepileptic drug therapy prevent the development of“chronic”epilepsy? Epilepsia 1996;37:701-8.

42. De Bittencourt , Bharucha et al, ILAE commission Report, Epilepsy in the tropics. Epidemiology, socioeconomic factors, epilepsia 1996, 37 : 1121-7

43. ILAE Commission Report. The epidemiology of the epilepsies : Epilepsia, 1997 : 38 : 614 -8.

44. Murthy JMK, Yangala, Etiological spectrum of symptomatic localization related epilepsies, JNS 1998; 158 : 65-70.

45. Sathishchandra, Kaliaperumal, Schoenberg et al, Hot water epilepsy from South India, Epilepsia 1988; 29; 52-6.

46. Koul R, Motta et al, Prevalence and pattern of epilepsy in rural Kashmir in India, Epilepsia 1988 ; 29 : 116-22.

47. Bharucha NE, Bharucha EP, Bhise, Schoenberg BS, Prevalence of epilepsy in the Parsis in Mumbay, Epilepsia 1988 ; 29: 111-5.

48. Mani KS, Rangan G, Srinivas HV, Kalyansundaram S, Narendran S, Reddy AK. The Yelendur study: A Community- based approach to epilepsy in rural South India- epidemiological aspects, Seizure 1998;7:281-8.

49. Commission on classification of ILAE, Proposal for revised International classification of epilepsies, epileptic syndrome, Epilepsia 1989; 30 : 389-99.

50. Hauser WA, Annegers JF, Rocca WA. Descriptive epidemiology of epilepsy: Contributions of population-based studies from Rochester, Minnesota. Mayo Clin Proc 1996;71:576-86.

51. Sridharan, Murthy, Prevalence and pattern of Epilepsy, Epilepsia 1999, 40; 631-6.

52. Murthy JMK, Yangala R, Mantha S. The syndromic classification of the International League Against Epilepsy. A hospital based study from South India. Epilepsia 1998;39:48-54.

53. Oka E, Istuda S, Outsuka I, Ohtahara S. Neuro-epidemiological study of childhood epilepsy by application of International classification of epilepsies and Epileptic Syndromes (ILAE,1989). Epilepsia 1995;36:658-61.

54. JainS.Padma MV.Puri A. Jyti, Maheswari MC. Occurrence of epilepsies in family Members of Indian probands with different epileptic syndromes. Epilepsia 1997;38:237-44.

55. Gandon I, Barato J, Aicardi J.Efficacite de la seanographie del'enfant. Ann Pediatr (Paris) 1983;30:112-5.

56. Viani F. Beghi E, Atza MG. Gulotta MP. Classification of epileptic syndromes. ages Advantages and limitations for evaluation of childhood epileptic syndromes in clinical practice. Epilepsia 1988;29:440-5.

57. Senanayake N.Roman G.Epidemiology of epilepsy in developing countries. *Bull WHO* 1993; 71L247-58.

58. Sander JWAS, Shorovon SD. Epidemiology of the epilepsies. J.Neurol Neurosurg Psychiatry 1996;61:431-43.

59. de Bittencourt PRM, Admolekum B.Barucha NE, et al: Epilepsy in the tropics 1: Epidemiology. Socioeconomics, risk factors and etiology. International League Against Epilepsy – Commission Report. Epilepsia 1996;37 : 1121-27.

60. de Bittencourt PRM, Admolekum B.Barucha NE, et al: Epilepsy in the tropics II: Clinical presentations, pathopysiology, immunologic diagnosis, economics and therapy. International League Against Epilepsy –Commission Report. Epilepsia 1996;37 : 1128-37.

61. Carpio A, Hauster WA. The distribution and etiology of epilepsy in the tropics of America. Rev Ecuat Neurol 1993: 2:137-45.

62. Murthy JMK, Yangala R.Etilogical spectrum of symptomatic localization-

related epilepsies: a study from South India. J Neurol Scie 1998;158: 65-70.

63. Bharucah NE, Bharucha EP, Bharucah AE, Bhise AV, Schoenberg BE.

Prevalance of epilepsy in the Parsi community of Bombay. Epilepsia 1988;29:111-5.

64. Koul R.Razdan S,Motta A.Prevalence and pattern of epilepsy in rural Kashmir, Epilepsia 1988;29:116-22.

65. Jallon P.Epilepsy in developing countries. ILAE workshop report. Epilepsia 1997;38:1143-51.

66. Das SK, Sanyal K.Neuroepidemiology of major neurological disorders in rural Bengal. Neurology India 1996;44 : 47-58.

67. Annegers JF. Hauser WA, Beghi E,Nocolosi A, Kurland LT. The riskunprovoked seizures after encephalitis and meningitis. Neurology 198 38:1407-10.

68. Dodge PR, Swartz MN. Bacterial meningitis : A review of selected aspects. N Engl J Med 1965 ; 272 : 954-60, 1003-10.

69. Marks DA, Kim J, Spencer DD, Spencer SS. Characteristics of intractable seizures following meningitis and encephalitis. Neurology 1992;42: 1513-8.

70. Pfister HW. Borasio GD, Dirnagl U, Bauer M, Einghaupl KM.Cerebrovascular complications of bacterial meningitis. Neurology 1992: 42:1497-1504.

71. Pomeroy SL, Holmes SJ, Dodge PR, Feigin RD. Seizure and other neurologic sequelae of bacterial meningitis in children. N Engl J Men 1990;323: 1651-7.

72. Tandon PN, Bhatia R, Bhargava S.Tuberculous meningitis. In Viken PJ.Bruyn GW, Klawans HL, eds. Amsterdam, Elsevier; 1988; 105-236.

73. Lorber J.Long-term followup of 100 children who recovered from tuberculous meningitis. Pediatrics 1961;28:778-82.

74. Donner M.Wasz-Hockert O. Late neurological sequelae of tuberculous meningitis. Acta Pediatr 1962:51 (Suppl 14):34-8.

75. Gowrie-DeviM, Ravi V, Shankar SK. Japanese encephalitis: an overview. In Clifford Rose F, ed, Recent advances in tropical neurology, Amsterdam: Elsevier Science BV, 1995; 217-35.

76. Venkatraman S.Neurocysticercosis scene in India. Prog Clin Neurosci 1989;5:297-314.

77. Singhal BS, Ladiwals U.SighalP. Neurocysticercosis in the Indian context with special reference to solitary parenchymal cyst. Neurology India 1997;45 :211-7

78. Mani AJ.Ramesh CK, Ahuja GK, Mani KS Cerebal cysticercosis presenting asepilepsy. Neurology India 1974; 22: 34-8.

79. Mahajan RC, Chopra JS. Cysticercosis amongst cases of epilepsy and intracranial space occupying lesion. In proceeding of National Seminar on Epilepsy, Bangalore 1975;95-7.

80. Singhal BS, Ladiwala NCC in India, Recent advances in Tropical Neurology Elsevier Science, 1995; 99-109.

81. Rajasekar, Etiology and Management SECTL, Act Neurology Scand, 1991; 84, 465-70

82. Rajasekar, Chacko, Clinico Radiological and pathological correlation in SCG, JNNP 1995, 59 ; 284-6.

83. Delbruto, Vadia, Proposal for diagnostic criteria for NCC, JNS, 1996, 143 ; 126.

84. Wadia RS, Makhale CN, Kelkar AB, Grant KB. Focal epilepsy in India with special reference of lesions showing ring or disc like enhancement on contrast CT.J Neurol Neurosurg Psychiatry 1987;50: 1298-1301.

85. Murthy JMK, Reddy YVS. Prognosis of epilepsy associated with single CT enhancing lesion: a long-term follow-up study. J Neurol Sci 1998. 159:151-5.

86. Rajshekhar V, Chandy MJ, Validation of diagnostic criteria for solitary cerebel cysticercus granuloma in patients presenting with seizures.Act Neurol Scand 1997;96 :76-81.

87. Caprio A, Escobar A, Hauser AW. Cysticercosis and epilepsy: a critical review. Epilepsia 1998;39 :1025-40.

88. Misra S,Verma R, Lekhra OP, Mishra NK. CT observations in partial seizures. Neurology India 1994;42:24-7.

89. Del Brutto OH, Santibanez R, Noba CA, Aguirre R, Diaz F. Epilepsy dev to neurocysticercosis : analysis of 203 patients. Neurology 1992;42 :389-92.

90. Murthy JMK, Reddy VS, Clinical characteristics, seizure spread patterns and prognosis of seizures associated with a single small cerebral calcific CT lesion. Seizure 1998;7: 153-7.

91. Mai NTH, Day NPJ, Choung LV, et al. Post-malaria neurologic syndrome.*Lancet 1996;*348:917-21.

92. Prabhakr S, Mathen DK. Subacute sclerosing panencephalitis, In Chopra JS,Sawhney IMS, eds, Neurology in tropics. N Delhi : BI Churchill Livignstone ; 1999;214-26.

93. Forsgren L.Bucht G. Erikson S, et al: Incidence and clinical characteristics of unprovoked seizures in adults- a prospective population based study Epilepsia 1996;37:224-9.

94. Nagraja D, Taly AB, Sarala Das. Puerperal cerebral venous thrombosis in India. Prog Clin Neurosc 1989;5:165-77.

95. Chopra JS, Benerjee AK, Primary intracranial sinovenous occlusion in youth and pregnancy. In Tolle JF, ed. *Handbook of Clinical Neurology,* Amsterdam, Elsevier. 1989:425-52.

96. Rao YP, Murthy JMK, Kishore LT.Prospective study of cerebral sinovenous thrombosis –value of CT scan and MR imaging, Neurology India 1996;44:152-8.

97. Canto C.Barringarrementeria F.Cerebral venous thrombosis associated with pregnancy and puerperium: review of 67 cases. Stroke 1993;24;1880-4.

98. Mani KS, Gopalakrishnan PD, Vyas JN, Pillai MS. Hot-water epilepsy: a peculiar type of reflex epilepsy. A preliminary report. Neurology India 1968:16:107-10.

99. Senanayake N.*Reflex Epilepsy.* Sri Lanka, Sarvodaya Vishava Lekha, 1998.

100. Devi TKM, Iyer GV.Pattern of triggering factors among patients with epilepsy in Kerala J Assoc Phys Ind 1985;33:513-5.

101. Chemburkar JA, Desai A. Reflex Epilepsy. Bull Jaslok Hospital 1977; 1:197-200.

102. Nagaraj D, Chand R P.Eating epilepsy.Clin Neurol Neurosurg 1984;86:95-9.

103. Ahuja GK, Pauranik A, Behari M, Prasad K. Eating epilepsy.J Neurol 1988;235:444-7.

104. Koul R, Kaul S, Razdan S. Eating epilepsy.Acta Neurol Scand 1989;80:78-80.

105. Mani KS, Rangan G. Epilepsy in the third world-Asian aspects. In: Dam M,Gram L, eds. Comprehensive epileptology. New York, Raven Press, 1990;781-93.

106. Maheswari MC.Compulsive waving to sun –a temporal lobe phenomenon.Neurology India 1978;26:123-5.

107. Iyer GV, Devi TKM, Rammanohar S. Heliotaxic epilepsy. J Assoc Phys Ind 1983;31: 283-5.

108. Iyer GV,RammanoharS, Devi TKM. Photosensitive epilepsy. Neurology India 1979;27:110-22.

109. Saleem SM, Thomas M, Jain S, Maheswari MC. Incidence of photosensitive epilepsy in unselected Indian epileptic population. Act Neurol Scand 1994;89:5-8.

110. Arts WF, Geerts AT, et al. The early prognosis of epilepsy in childhood: the prediction of a poor outcome. The Dutch study of epilepsy in childhood. Epilesia 1999;40(6):726-734.

111. Berg AT, Shinnar S, et al. Defining early seizure outcomes in pediatric epilepsy: the good, the bad and the in-between. Epilepsy Res 2001;43(1);75-84.

112. Shinnar S.Berg AT, et al. Predictors of multiple seizures in a cohort of children prospectively followed from the time of their first unprovoked seizure [see comment]. Ann Neurol 2000; 48(2);140-147.

113. Berg AT, Levy SR, et al. Predictors of intractable epilepsy in childhood; a case-control study. Epilepsia 1996;37(1): 24-30.

114. Casetta 1, Granieri E, et al.Early predictors of intractability in childhood epilepsy: a community-based case-control study in Copparo, Italy. Acta Neurol Scand 1999;99(6):137-139.

115. Rajadyaksha Shah, Kanhere, Naik, Mansapdar et al, Pattern and prevalence of Epilepsy and Epileptic syndromes in childhood in India, Epilepsy, The Indian perspective Chapter 3, page 37-60.

116. Wael Hayel et al PJMS (Pakistan Journal) vol. 22, Page 1-7 Pattern of Epilepsy in less than 2 years of age.

117. Afzal et al – J. College of Physicians and Surgeons of Pakistan, Epidemiology and etiological profile of Epilepsy in Pakistan.

ABBREVIATIONS USED

EEG	–	ELECTRO ENCEPHALOGRAM
CT	–	COMPUTED TOMOGRAPHY
ILAE	–	INTERNATIONAL LEAGUE AGAINST EPILEPSY
ICSE	–	INTERNATIONAL CLASSIFICATION OF SEIZURE EPIDEMIOLOGY
WS	–	WEST SYNDROME
LGS	–	LENNOX GESTAUT SYNDROME
JE	–	JAPPANESE ENCEPHALITIS
PMS	–	POST MENINGITIC SEQUALE
PES	–	POST ENCEPHALITIC SEQUALE
NCC	–	NEURO CYSTI CERIOSIS
NFLE	–	NOCTURNAL FRONTAL LOBE SPILEPSY
SCTEL	–	SINGLE CONTRAST ENHANCING LESION
NMD	–	NEURONAL MIGRATION DISORDER
ACC	–	ABSENCE OF CORPUS COLLOSUM
DCC	–	DYSGENESIS OF CORPUS COLLOSUM
TS	–	TUBEROUS SCLEROSIS
SWS	–	STURUE WEBER SYNDROME
HIE	–	HYPOXIC ISCHEMIC ENCEPHALOPATHHY
MR	–	MENTAL PETARDATION
JME	–	JUUENILE MYOCLONIC EPILEPSY
CVA	–	CEREBRO VASCULAR ACCIDENT
CAS	–	CHILD HOOD ABSENCE SEIZURES
Rt FOCAL	–	RIGHT FOCAL
Lt FOCAL	–	LEFT FOCAL
SW	–	SHARP WAVES

SS	–	SPIKES AND SHARPWAVES
PS	–	POLY SPIKES
CD	–	CEREBRAL DYSFUNCTION
BGA	–	BACKGROUND ABNORMALITY
BGS	–	BACKGROUND SLOWING
HA	–	HYPSARRHYTHMIA
FD	–	FOCAL DISCHARGES
CTS	–	CENTREO TEMPORAL SPIKES
N	–	NORMAL
GO	–	GOOD OUT COME
IO	–	INTERMEDIATE OUTCOME
BO	–	BAD OUT COME
M	–	MALE
F	–	FEMALE

Reg.No	Name	Sex	Age	Age of Onset	Clinical Type	EEG	CT.Scan Finding/ Etiology	Total Episodes	Follow Up	Outcome
126/04	Nagarajan	M	4	4	GTCS	BGS +SS	Lissencephaly	4	2Y	G.O
265/04	Balamurugan	M	9	5Y	GTCS	S.W.	Idiopathic	2	2Y	G.O
19/02	Vigneshkumar	M	8	5Y	GTCS	S.S	Idiopathic	2	2Y	G.O
180/05	Kannan	M	8	1Y	GTCS	S.W	P.M.S	4	2Y	G.O
137/05	Karthick	M	5	3Y	GTCS	S.S.	Idiopathic	3	2Y	G.O
79/05	Sangesh	M	9	2Y	GTCS	S.S	Idiopathic	3	2Y	G.O
127/05	Dinesh Kumar	M	10	5	LT Focal	C.T.S	Idiopathic	10	2Y	G.O
229/03	Trisha	F	¾	1 ½	LT Focal	RT FD	Idiopathic	2	2Y	G.O
211/03	Punitha Valli	F	6	3	Rt Focal	LT FD	Idiopathic	2	2Y	G.O
152/02	Ramar	M	3	2 ½	Rt Focal	S.S	Idiopathic	2	2Y	G.O
210/03	Sivalingaraja	M	10	8	Rt Focal	Lt FD	Calified Granuloma	2	2Y	G.O
104/03	Jayakumar	M	3½	2	Rt Focal	S.S	Tuberculoma	2	2Y	G.O
272/03	Saravana Kumar	M	8 ½	7Y	Lt Focal	Rt F.D	Tuberculoma	2	2Y	G.O
91/03	Anitha	F	6	3 ½	Rt Focal	F.D	NCC	2	2Y	G.O
325/04	Senthamil Selvi	F	9	8	Rt Focal	S.W	Tuberculoma	2	2Y	G.O
143/03	Vinodhini	F	3	1	Rt Focal	S.W	ASPHYXIA	2	2Y	G.O
55/04	Pooja	F	3 ½	2	Rt Focal	S.S	ASPHYXIA	2	2Y	G.O
10/98	Selva Kumar	M	10	2	Rt Focal	S.W	Tuberculoma	3	2Y	G.O
30/03	Muthu Karuppu	M	5	1½	Rt Focal	S.W	NCC	2	2Y	G.O
142/01	Hariharan	M	4	6/12M	Rt Focal	B.G.S + S.W	NFLE	4	2Y	G.O
210/04	Pandi	M	3 ½	6/12M	Rt Focal	S.W	ASPHYXIA	2	2Y	G.O
134/04	Srinivasan	M	5	4	Rt Focal	N	Tuberculoma	2	2Y	G.O

122/04	Gayathri	F	11	10	Rt Focal	S.W	NCC	2	2Y	G.O
152/04	Anandhi	F	8	7	Rt Focal	C.T.S	Idiopathic	4/D	2Y	I.O
291/04	Priya	F	10	9	Rt Focal	S.S	Idiopathic	3	2Y	G.O
363/05	Soundareswari	F	7	6 ½	Rt Focal	F.D	NCC	2	< IY	--
258/05	Hakkim	M	7	1	GTCS	N	Granuloma	3	< IY	--
349/05	Nitin	M	2½	2 ½	GTCS	S.W	Idiopathic	3	6M	-
193/05	BHRamman	M	7	6 ½	RT Focal	FD	NCC	5	6M	-
311/05	Kowsalya	F	9	9	RT Focal	S.W	Clasified Granuloma	2	6M	-
257/05	Balamurugan	M	7 ½	7	RT Focal	S.S	Idiopathic	6	6M	-
256/05	Ranjitha	F	10	9 ½	RT Focal	S.W	Clasified Granuloma	2	6M	-
353/05	Alagarsamy	M	10	10	RT Focal	N	Idiopathic	2	6M	-
345/05	Veerapandi	M	8 /12	8/12M	GTCS	S.S	Asphyxia	2	6M	-
223/05	Nagaraj	M	5 ½	5	RT Focal	S.W	Tuberculoma	8	6M	-
125/05	Mehboob	M	11	8	RT Focal	S.S	NLC	2	6M	-
87/05	Selvakumar	M	11		RT Focal	S.W	Idiopathic	7	<1Y	-
184/05	Pandiselvi	F	1 ¼	5/12M	Myoclonus	C.D	PMS	6	6M	-
22/04	Karthik	M	1 ½	2/12	GTCS	S.W	Idiopathic	2	<1Y	-
36/97	Ramamoorthy	M	9	2/12M	Tonic	S.S	Asphyxia	2	6M	-
106/05	Jeyavel	M	1 ¼	1 ¼	Tonic	S.S	Asphyxia	2	6M	-
127/04	Kalaivani	F	5	41/2	GTCS	C.D	P.M.S	2	6M	-
218/04	Ajith	M	8	8	GTCS	S.W	Idiopathic	2	6M	-
2522/05	Zaheer	M	1 ½	10/12	GTCS	B.G.S +S.S	P.E.S	4	6M	-
114/05	Vayakattu Samy	M	10/12	8/12	Tonic	N	Idiopathic Reflexepilepsy	2	6M	-
261/04	Venkatesh	M	1 ¾	8/12	GTCS	B.G.A	Asphyxia	2	6M	-
148/05	Lokesh	M	1	10/12	GTCS	S.W	Asphyxia	2	6M	-
135/05	Kamselvi	F	4/12	3/12	Tonic	N	Asphyxia	2	6M	-

403982	Sivakishor	M	5 ½	5 ¼	Absence	S.S	Idiopathic	5/D	6M	-
264/05	MadhanRaj	M	4 ½	4	Myoclonos	P.S	Schizencephaly	5/D	6M	-
26/05	Meeradevi	F	9	8 ½	RT Focal	S.W	Idiopathic	4	6M	-
110/04	Subitha	F	7	7	LT Focal	F.D	Tuberculoma	2	6M	-
138/05	Andiambalam	M	1 ¾	1	LT Focal	S.S	Porencephaly (Asphyxia)	1/M	6M	-
74/04	Prema	F	10	10	RT Focal	N	Tuberculoma	2	6M	-
103/05	Thangapandiyan	M	4	3 ¾	RT Focal	S.W	Asphyxia	2	6M	-
227/03	Gayathri	F	9	7	RT Focal	S.W	Tuberculoma	2	<1Y	-
102/05	Vignesh	M	10	10	RT Focal	S.S	NCC	2	<1Y	-
182/05	Thavamani	M	10	9	Lt Focal	S.W	Tuberculoma	2	<1Y	-
12/05	Venkatesh	M	5	4 ½	Rt Focal	S.W	Tuberculoma	2	<1Y	-
308/04	Pavithra	F	7	6 ½	RT Focal	F.D	Tuberculoma	2	<1Y	-
326/04	Alagumurugan	M	1	2/12	RT Focal	S.S	Asphyxia	2/D	<1Y	-
65/05	Jayeela	F	10	10	RT Focal	S.W	NCC	2	<1Y	-
108/05	Anand	M	6	6	Lt Focal	FD	Tuberculoma	5	<1Y	-
104/03	Ulagammal	F	1	2/12	Lt Focal	FD	Asphyxia	2	<1Y	-
658/80	Buvaneshwari	F	2 ½	1/12	Rt Focal	S.W	Asphyxia	3	<1Y	-
90/05	Arun	M	7/12	3/12	Lt Focal	SS	Asphyxia	2/D	6M	-
27/05	Sathyan	M	5/12	5	Lt Focal	FD	PES	15	6M	-
128/04	Martyn	M	3	3	Lt Focal	S.W	Idiopathic	3	6M	-
136/04	Jothi	F	9	9	Lt Focal	CD	Asphyxia	2	6M	-
264/04	Mohamed Haris	M	2 ½	2 ½	RT Focal	LTS	Idiopathic	3	<1Y	-
98/05	Hariharan	M	6	6	RT Focal	S.S	Idiopathic	2	6M	-
30/06	Logakeerthi	F	8/12	3/12	Myoclonus	B.G.A	Lissencephaly	3/D	6M	-
217/04	Lathish	M	1	3 ½	GTCS	S.S	SWS	2	<1Y	-
110/04	Subash	M	4 ½	3 ½	GTCS	S.S	Torch	2	<1Y	-

42/04	Praveen	M	10	2	Lt Focal	S.W	Idiopathic	1/M	<1Y	-
27/04	Sathya	M	5 ¾	3 ½	Lt Focal	S.S	P.E.S	2/M	2Y	B.O
116/04	Paramesh	M	3	1 ½	Lt Focal	S.S	Asphyxia	2/M	2Y	B.O
10/04	Mookammal	F	2	1	GTCS	S.S	Asphyxia	2/D	2Y	B.O
421/2K	Saranya	F	9	3 ½	Myoclonus	S.S	P.M.S	2/D	2Y	B.O
31/03	Sathya Raj	M	5 ½	3	Lt Focal	S.S	P.M.S	1/M	2Y	B.O
33/02	Alaudeen	M	7	9/12	Myoclonus	B.G.S	Asphyxia	1/M	2Y	B.O
90/2K	Aathilingam	M	8	3/12	Myoclonus	S.S	Asphyxia	4/WK	2Y	B.O
32/03	Ebinesar	M	1 ½	1/12M	Tonic	S.W	Asphyxia	2/M	2Y	B.O
60/04	Salomi	F	6	1 ½	GTCS	C.D	Asphyxia	2/M	2Y	B.O
128/96	Praveen Kumar	M	10	2 ½	Myoclonus	P.S	Asphyxia	4/D	2Y	B.O
169/04	Sebastian	M	6	1/12	Myoclonus	BGS	Asphyxia	1/D	2Y	B.O
36/98	Raman	M	9	1 ½	GTCS	S.W	Asphyxia	1/M	2Y	B.O
320/96	Ramkumar	M	12	5	Myoclonus	P.S	P.M.S	1O/D	2Y	B.O
108/03	Santosh	M	8	1 ½	GTCS	C.D	Lissencephaly	3/D	2Y	B.O
93/02	Uma	F	7	3	GTCS	S.S	Asphyxia	1/D	2Y	B.O
74/96	Prasanth	M	10	11/12	RT Focal	F.D	Asphyxia	2/M	2Y	B.O
71/98	Anandhraj	M	10	3 ½	RT Focal	F.D	P.E.S	1/M	2Y	B.O
127/03	Dinesh Kumar	M	11	1	RT Focal	S.W	Porencephaly(Asphyxia)	4/M	2Y	B.O
64/03	Dinesh Kumar	M	12	5	RT Focal	S.W	Asphyxia	2/M	2Y	B.O
73/98	Sabeena	F	10	3	RT Focal	S.W	Asphyxia	1/D	2Y	B.O
160/01	Sabari	M	5 ½	1 ½	RT Focal	C.D	Asphyxia	2/M	2Y	B.O
262/99	Dinesh	M	12	5	RT Focal	S.W	C.V.A	1/M	2Y	B.O
204/03	Sathish Kumar	M	12	9	RT Focal	S.W	Tuberculoma	2/M	2Y	B.O
3/03	Sudarsan	M	3 ½	1 ½	Myoclonus	S.S	Torch	20/D	2Y	B.O
91/04	Navina	F	8	9/12	Rt Focal	S.S	P.M.S	2/NK	2Y	B.O
52/06	Mahalaxmi	F	1 ½	1 ½	GTCS	S.W	Idiopathic	3	6M	-

74/06	Rajasekar	M	10	10	GTCS	S.W	Idiopathic	2	6M	-
48/2k	Sundarakumar	M	6	6/12	Rt Focal	S.S	Idiopathic	1/M	2Y	I.O
236/05	Muthumari	M	1	10/12	GTCS	S.W	Asphyxia	6	6M	-
246/04	Sathyaraj	M	4 ½	7/12	Myoclonus	S.W	P.M.S	3/D	2Y	B.O
215/05	Lingeswari	F	9/12	45D	GTCS	C.D	Idiopathic	4	6M	-
274/04	Gomathi	F	4	6/12M	GTCS	S.W	Idiopathic	4	2Y	I.O
48/06	IShwarya	F	10	10	GTCS	S.W	Idiopathic	2	6M	-
212/04	Ajith	M	8	6	Rt Focal	S.W	Idiopathic	2	2Y	G.O
47/06	Durgadevi	F	2 ½	1 ½	GTCS	S.S	Asphyxia	4	6M	-
40/04	Ram mohan raj	M	12	10	GTCS	S.W	Tuberculoma	2	2Y	G.O
301/05	Peivendar	M	1 ½	1	GTCS	C.D	Asphyxia	2	6M	-
38/03	Alagar	M	5	4	GTCS	C.D	Asphyxia	2	<1Y	-
172/04	Ashok Kumar	M	11	9	GTCS	N	Idiopathic	4	2Y	I.O
149/015	Jallikattu	M	1 ½	1	GTCS	S.W	Idiopathic	2	6M	-
301/04	Prakash	M	7	4	GTCS	S.W	Idiopathic	4	2Y	I.O
224/04	Vijay Baskar	M	3	6/12	GTCS	S.S	Asphyxia	6/D	1 ½	I.O
124/04	Sivasakthi	M	31/2	2 ¾	GTCS	S.S	Asphyxia	5	6M	-
284/04	Ramyakrishnan	F	21/2	2 ½	GTCS	C.D	Asphyxia	3	6M	-
15/06	Mary	F	9	8 ½	GTCS	S.W	NCC	2	6M	-
243/05	Kumar	M	11/2	1	GTCS	S.S	Asphyxia	2	6M	-
69/06	Umashankar	M	12	11	Lt FocaL	F.D	NCC	3	<1Y	-
29/03	Barathy	M	2	5/12	GTCS	N	Idiopathic	2	2Y	G.O
139/05	Prabahar	M	9	9	GTCS	N	Idiopathic	2	6M	-
316/04	Ajay	M	1 ½	1	GTCS	N	Idiopathic	2	6M	-
71/04	Suryaraj	M	2	1	GTCS	S.W	Idiopathic	2	<1Y	-
111/99	Muthumari	F	12	3	GTCS	S.S	P.M.S	2/D	2Y	B.O
61/05	Fatima	F	6	6/12	GTCS	S.W	Asphyxia	15	2Y	I.O
282/04	Ramya	F	9	8	Lt FocaL	N	Idiopathic	3	2Y	I.O

167/05	Murshida	F	12	10	Rt FocaL	S.S	Tuberculoma	5	2Y	I.O
49/99	Selvi	F	9	3	Lt FocaL	N	Tuberculoma	10	2Y	I.O
81/98	Premkumar	M	12	3	Rt FocaL	S.W	Tuberculoma	10	2Y	I.O
223/03	Vijay Babu	M	12	4	Lt FocaL	S.S	Tuberculoma	12	2Y	I.O
123/02	Veeramani	M	11	3	Rt FocaL	S.W	NCC	5	2Y	I.O
19/04	Sivasankari	F	11	10	Lt FocaL	N	Tuberculoma	4	2Y	I.O
56/05	Sivaroopan	M	1 ¼	2m	Tonic	S.S	Asphyxia	2	1Y	I.O
108/04	Manibarathi	M	5	2 ½	Tonic	S.S	Asphyxia	4	2y	I.O
182/05	Senthoor pandi	M	11	8	Rt Focal	C.T.S	Idiopathic	2/m	1y	I.O
97/05	Vijayadarshini	F	4	2 ½	Rt Focal	S.W	Asphyxia	10	1y	I.O
38/06	Divya	F	4	3 ½	Rt Focal	S.S	P.M.S	3/D	2y	I.O
99/04	Jeeva	M	3	2	GTCS	S.W	Idiopathic	3	1 ½ y	I.O
248/04	Ramar	M	1 ½	3/12	GTCS	S.S	Asphyxia	2/M	1 ½ y	I.O
337/04	Thavaselvi	F	1 ½	6/12	Myoclonus	C.D	P.M.S	2	1 ½ y	I.O
198/03	Anitha	F	2 ½	9/12	Myoclonus	H.A	P.E.S	2/D	2Y	I.O
320/04	Sivabarathi	M	2	1	GTCS	S.S	Idiopathic	2	1 ½ y	I.O
300/04	Yuvraj	M	4	6/12	Myoclonus	S.S	Asphyxia	8	1 ½ y	I.O
120/03	Karuppiah	M	8	5	GTCS	N	Asphyxia	2	2y	I.O
32/96	Balanandini	F	12	4	GTCS	S.W	Idiopathic	10/Y	2y	I.O
150/04	Rajkumar	M	9	8	GTCS	S.S	Idiopathic	2	1 ½ y	I.O
52/04	Manimegala	F	7	3	Atonic	S.W	Idiopathic	6	1 ½ y	I.O
183/04	Anitha	F	10	9	GTCS	S.S	Idiopathic	7	1 ½ y	I.O
280/04	Valarmathy	F	5	4	GTCS	S.S	DCC	4	1 ½ y	I.O
241/04	Pandi	M	2	1	GTCS	S.S	Asphyxia	2	1y	I.O
130/02	Nandhini	F	3	2	GTCS	S.W	Idiopathic	5	2y	I.O
99/04	Jeeva	M	3	2	GTCS	S.W	Idiopathic	10	1 ½ Y	I.O
160/04	Ranjitham	F	11	10	GTCS	S.W	Idiopathic	2	1 ½ Y	I.O
152/04	Anandhi	F	8	7 ½	GTCS	N	Idiopathic	10	1 Y	I.O

35/05	Ranjani	F	1 ½	1/12m	GTCS	S.S	Asphyxia	1/WK	1 ½ Y	I.O
204/04	Manoj	M	4	3	GTCS	S.S	Asphyxia	3	1 ½ Y	I.O
135/03	Vinodh	M	12	11	GTCS	S.S	Idiopathic	5	1Y	I.O
192/03	Meenakshi	F	7	6	GTCS	S.S	Asphyxia	2	2y	I.O
270/04	Soundarya	F	4	3	Lt FocaL	C.T.S	Idiopathic	3	1Y	I.O
45/02	Ayyanar	M	4	3/12	Rt Focal	S.W	Asphyxia	10	2y	I.O
101/03	Manikandan	M	9	8	Rt Focal	S.W	NCC	3	2y	I.O
100/03	Muthukaruppu	M	6	1	Rt Focal	S.W	Asphyxia	2/Y	2y	I.O
211/04	Sakthibala	M	4	3	Rt Focal	S.W	NCC	2	1 ½ Y	I.O
178/03	Vijay	M	5	3	Lt FocaL	S.S	Tuberculoma	12	2y	I.O
47/03	Soundarya	F	7	5	Lt FocaL	S.W	Tuberculoma	15	2y	I.O
67/03	Vijay	M	6	5 ½	Lt FocaL	F.D	P.E.S	2/WK	2y	I.O
39/98	Shenbagam	F	8	3/12	Rt Focal	C.D	Asphyxia	6	2y	I.O
240/05	Menaka	F	4 ½	3	Rt Focal	S.W	Idiopathic	3	1 ½ Y	I.O
76/05	Muthuveeran	M	1 ½	6/12	Myoclonus	S.S	Idiopathic	3/D	1 Y	I.O
241/04	Pandi	M	1 ½	5/12	GTCS	S.S	Asphyxia	3/M	1 ½	I.O
62/03	Pandeswari	F	3	5/12	GTCS	S.S	Asphyxia	2	2Y	G.O
30/96	Thowbeek	M	10	4/12	GTCS	B.G.S	Asphyxia	4/WK	2Y	B.O
67/04	Sanjaykumar	M	4	1	GTCS	S.S	Asphyxia	3	1 ½	G.O
121/04	Kaliammal	F	5	7/12	GTCS	S.W	Asphyxia	2	2Y	G.O
74/01	Karthik	M	5	6/12	Rt Focal	S.W	Asphyxia	5	2Y	G.O
370/04	Sivam	M	7/12	3/12	GTCS	S.S	Asphyxia	4	<1Y	-
261/05	Harshidabanu	F	7	7	Rt Focal	N	Tuberculoma	2	<1Y	-
337/05	Naveen	M	5	1 ½	GTCS	S.W	ACC	2	2Y	G.O
271/03	Vani	F	5	3	Rt Focal	N	Tuberous Sclerosis	2	2Y	G.O
96/04	Kousalya	F	9	8	Rt Focal	S.S	Tuberculoma	2	1 ½	G.O
4/04	Arunjothi	F	6	1	GTCS	S.W	Idiopathic	10	1 ½	I.O
123/03	Marudupandi	M	7	2	Rt Focal	S.S	Asphyxia	5	2Y	G.O

320/96	Ramkumar	M	12	4	Rt Focal	N	Tuberculoma	10	2Y	G.O
102/01	Sathish kumar	M	7	3	Rt Focal	F.D	Tuberculoma	4	2Y	G.O
201/02	Santosh Kumar	M	6	3	Rt Focal	F.D	Tuberculoma	3	2Y	G.O
346/02	Priya	F	3 ½	1	GTCS	S.S	Asphyxia	2	2Y	G.O
135/04	Vinodh	M	12	6	GTCS	S.W	Idiopathic	2	1 ½ Y	G.O
168/03	Barathi	M	2 ½	2 ½	GTCS	S.S	Idiopathic	2	2Y	G.O
3107	Prabu	M	3 ½	3 ½	GTCS	S.S	Idiopathic	8	2Y	G.O
137/2K	Ganesha	M	2 ½	2 ½	ABSENCE	SPIKES C3HZ	Idiopathic	10	2Y	G.O
86/01	Senthil	M	10	10	GTCS	S.S	Idiopathic	2	2Y	G.O
73/03	Ajisha	F	4	4	GTCS	S.W	Idiopathic	2	2Y	G.O
274/02	Saravanan	M	1	1	TONIC	B.G.A	P.E.S	2	2Y	G.O
5049	Kannan	M	6	6	GTCS	S.S	Idiopathic	2	2Y	G.O
94/04	Karthiga	F	3	3	GTCS	S.S	Idiopathic	2	1 ½ y	G.O
104/05	Roopan	M	6/12	6/12	GTCS	S.S	Asphyxia	2	1y	G.O
55/02	Karthigai Kumar	M	3	3	GTCS	S.S	Asphyxia	6	2Y	G.O